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# UPPER-RESPIRATORY INFECTION AS A FACTOR INFLUENCING SUSCEPTIBILITY TO POLIOMYELITIS*

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BOSTON

POLIOMYELITIS is now definitely fixed as predominantly a subclinical immunizing infection in which paralysis is the exceptional event.[1-3] Ever since the 1916 epidemic, evidence has slowly accumulated that factors other than exposure to the virus,—[4, 5] factors as diverse as tonsillectomy,[6-8] pregnancy[9] and endocrine function,[10-11] influence the paralytic manifestations of poliomyelitis. Likewise clinical and laboratory evidence suggests that overexertion and fatigue, chilling or overheating and preceding illness may act during the prodromal period as trigger mechanisms to precipitate paralysis.[12-16] Recently McCloskey[17] and Geffen[18] have found that a significant number of paralytic cases follow injection of diphtheria toxin and pertussis vaccine by the incubation interval, as in the case of tonsillectomy. A high degree of localization of paralysis in the extremity in which the injection is made gives added significance to the fact.[19]

These observations suggest the hypothesis that naturally occurring infectious agents may influence the development of the clinical manifestations of poliomyelitis and prompt a re-examination of epidemiologic material assembled in 1936 but not heretofore published. The data relate to a school outbreak of poliomyelitis characterized by an unusually high attack rate among the total number of boys who could possibly have been exposed to the virus. This outbreak was shortly preceded by a significantly high incidence of upper-respiratory infections in the school.

## EPIDEMIOLOGY

### *Poliomyelitis in Southboro, Massachusetts*

In the latter half of May, 1936, with only 8 antecedent cases reported in the state, an explosive outbreak of poliomyelitis involving 22 (11 per cent) of 190 boys occurred at St. Mark's School in Southboro, Massachusetts (Fig. 1). Otherwise, cases of poliomyelitis in Massachusetts (population then 4,425,000 persons) were few, sporadic and widely distributed throughout the year (Fig. 2). This was the lowest reported incidence on record in the Commonwealth in contrast to the preceding year,

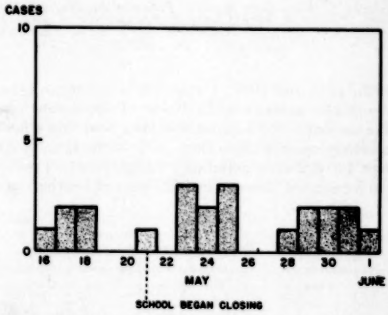


FIGURE 1. *Twenty-two Cases of Poliomyelitis, St. Mark's School, May, 1936, by Day of Onset of Signs of Meningeal Irritation.*

1935, when the number reported reached the third highest figure in the history of the state, — 1390 (Table 1). A spot map clearly indicates the unusual concentration of cases in Southboro, numbering some 2100 inhabitants, especially when compared with cities the size of Boston (2 cases), Cambridge (3 cases), Worcester (3 cases) and Lowell (4 cases) (Fig. 3).

Southboro is a small farming and residential town located about 25 miles west of Boston and covering approximately 25 square miles. A dozen buildings on either side of the main street comprise its center, from which St. Mark's School is only a few hundred yards removed and set off by athletic fields. The general plan of the main building is that of an H

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with attached wings — headmaster's wing, chapel, library and kitchen.

During the thirty-year period from January 1, 1918, to January 1, 1948, poliomyelitis was reported to the Southboro Board of Health in only five years other than 1936: in 1920, 5 cases; in 1923,

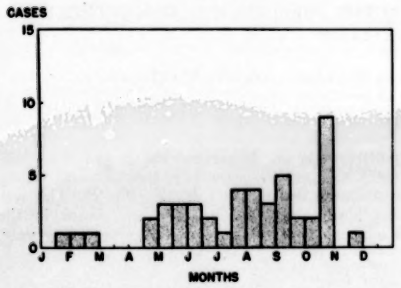


FIGURE 2. *Forty-Seven Cases of Poliomyelitis, Massachusetts, 1936, by Month of Onset.*

1926, 1946 and 1947, 1 case. It is interesting that the small outbreak in 1920 was of the family type like the one to be described — for a boarding school, epidemiologically speaking, is a large family, except for different hereditary composition. Four of the 5 reported illnesses in 1920 were of brothers and

TABLE 1. *Poliomyelitis in Massachusetts.*

| YEAR | POPULATION | CASES | RATE PER 100 000 PERSONS |
|---|---|---|---|
| 1916 | 3,719,000 | 1927 | 51.8 |
| 1931 | 4,280,000 | 1428 | 33.3 |
| 1935 | 4,361,570 | 1390 | 31.9 |
| 1936 | 4,425,000 | 60 | .1 |

sisters of a single family. Dates of onset were given as August 28 and September 8, 12 and 13 and the ages as 7, 4, 10 and 5 years, respectively.

In 1936 the school numbered among its students 190 boys, 12 to 19 years of age, distributed into six forms (Table 2). They were housed in the main building, attended chapel together and ate in a common dining room. During the period of May 16 through June 2 (17 days), 22 boys developed poliomyelitis. Thirteen had paresis or paralysis to a greater or lesser extent; seven had permanent sequelae; and one died. The virus of poliomyelitis was recovered from tissues of this patient.[20]

No other cases occurred in the town of Southboro. Three principal groups with no cases included the 250 students in the local high school, 125 in another boarding school and about 20 St. Mark's faculty children under 18 years of age. Both other school buildings are within 400 yards of the primary focus, and students came into random contact with each other at the community sandwich shop, restaurant, drugstore, barber shop and tailoring shop.

Investigation of the circumstances of the outbreak was undertaken jointly by representatives of the Harvard Infantile Paralysis Commission and of the United States Public Health Service. The extensive data compiled by these agencies at the time and since supplemented by study of clinical and hospital records are the basis of this report.

*Respiratory Infections Prior to Outbreak of Poliomyelitis*

During the months of April and May, 1936, 52 boys were admitted to the school infirmary with

FIGURE 3. *Geographical Distribution of 22 Cases of Poliomyelitis Originating in Southboro and 47 Cases Reported from Other Localities in Massachusetts during 1936.*

upper-respiratory infections — an attack rate of about 27 per cent. In general those admitted in April (13 of 17) ran temperatures under 100°F., whereas those of May (21 of 35) had fever of more than 100°F. Clinically, these patients appeared to

TABLE 2. *Classification of 190 Students by Age and Form, St. Mark's School, May, 1936.*

| AGE | FORM | | | | | | TOTAL | |
|---|---|---|---|---|---|---|---|---|
| | I | II | III | IV | V | VI | NO. | PERCENTAGE |
| 12.5 | 6 | 0 | 0 | 0 | 0 | 0 | 6 | 3.1 |
| 13.5 | 10 | 10 | 1 | 0 | 0 | 0 | 21 | 11.1 |
| 14.5 | 2 | 14 | 10 | 0 | 0 | 0 | 26 | 13.7 |
| 15.5 | 0 | 4 | 21 | 16 | 0 | 1 | 42 | 22.1 |
| 16.5 | 0 | 1 | 8 | 22 | 5 | 0 | 36 | 19.0 |
| 17.5 | 0 | 0 | 0 | 0 | 24 | 7 | 31 | 16.3 |
| 18.5 | 0 | 0 | 0 | 0 | 6 | 16 | 22 | 11.6 |
| 19.5 | 0 | 0 | 0 | 0 | 0 | 6 | 6 | 3.1 |
| All ages | 18 | 29 | 40 | 38 | 35 | 30 | 190 | 100.0 |

have the purulent types of pharyngitis, tonsillitis, sinusitis or otitis media characteristically associated with pyogenic bacteria. In 35 cases the chief complaint was sore throat; in others simply a "cold," a "sinus" or an earache. Often such complaints were multiple. Only 2 throat cultures are recorded. From

one, *Staphylococcus aureus* was recovered; from the other, a mixture of bacteria: pneumococcus, Types 3 and 4, *Streptococcus viridans* and *hemolyticus*, and *Staphylococcus albus* and *aureus*.

The sweep of these mixed infections in relation to the subsequently occurring outbreak of poliomyelitis is shown in Figure 4. Cases are arranged in

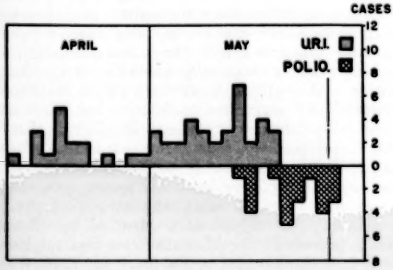


FIGURE 4. *Fifty-Two Boys Hospitalized for Acute Upper-Respiratory Infection and 22 Boys with Poliomyelitis, Arranged by Two-Day Intervals, April and May, 1936.*

two-day intervals by dates of admission to the hospital.

That 52 admissions during the spring term of 1936 is an unusual incidence of respiratory infections at St. Mark's School is indicated by comparison with the spring terms over the ten-year period from 1939–1948 inclusive (Table 3). As the same 2

TABLE 3. *Admissions to Infirmary for Acute Upper-Respiratory Infections* Spring Term, St. Mark's School, 1939-1948.*

| YEAR | POPULATION | ADMISSIONS | RATE PER CENT |
|---|---|---|---|
| 1939 | 197 | 22 | 11.2 |
| 1940 | 195 | 20 | 10.3 |
| 1941 | 195 | 12 | 6.2 |
| 1942 | 191 | 11 | 5.6 |
| 1943 | 179 | 15 | 8.4 |
| 1944 | 181 | 11 | 6.1 |
| 1945 | 181 | 7 | 3.9 |
| 1946 | 193 | 15 | 7.8 |
| 1947 | 205 | 28 | 13.7 |
| 1948 | 205 | 16 | 7.8 |
| Average 1939–1948, inclusive | 191 | 15.7 | 8.2 |

*Sore throats, pharyngitis, tonsillitis, colds, coughs, bronchitis, otitis (exanthemata or mumps not included).

nurses acted as admitting officers for over fifteen years there is no reason to suppose criteria for admission were any different in 1936 from those of subsequent years. Boys with mild symptoms and no fever are treated in the dispensary as ambulatory patients. Those with fever or prostration are admitted to hospital. During the ten-year period specified, average admissions were 16 per spring term. The figure varies from seven in 1945 to 28 in the spring of 1947, when complement-fixation tests on paired serums indicated that influenza was prevalent.

Closely following the wave of respiratory infections, 5 cases of poliomyelitis were identified on May 16, 17 and 18. The remaining 17 cases developed between May 21 and June 2. Eleven of these boys had been withdrawn from school about May 20. During the period of May 1 through May 20, 9 (41 per cent) of the boys who later developed poliomyelitis had respiratory infections. During

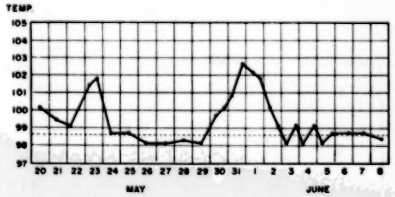


FIGURE 5. *Temperature Chart of Patient Admitted to School Infirmary With Upper-Respiratory Infection Followed by Poliomyelitis 11 Days Later.*

the same period only 30 (19 per cent) of others in the school had such illness. In all, 10 students contracted poliomyelitis who had had a preceding upper-respiratory infection during the spring term

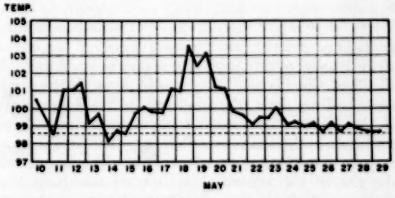


FIGURE 6. *Temperature Chart of Boy Going Directly to Infirmary from Swimming Pool with Sore Throat on May 10, 1936 (Stiffness of Neck Developed May 17th).*

of 1936. The interval between onsets of the two types of illness ranged from four to twenty-two days and averaged about nine and a half days. This relation is shown by the temperature charts of 2 of the patients in Figures 5 and 6 and for the group in Figure 7. Five of the 10 exhibited signs of bulbar involvement manifested by ocular, facial or faucial weakness. No others are recorded as having shown signs of bulbar involvement. These distributions carry a definite implication that upper-respiratory infection was one of several factors in the determina-

tion of clinical disease during this outbreak characterized by unusually high frequency of clinical manifestations among the population at risk—even if all 190 boys were infected with the virus of poliomyelitis.

### Distribution of Cases

Table 4 shows the distribution of poliomyelitis and respiratory infections by ages and forms. The sixth form, comprising over 16 per cent of the school population, escaped manifestations of poliomyelitis and had the lowest rate for upper-respiratory infections. That sixth-form members, being the

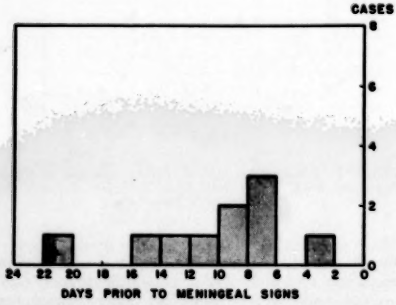


Figure 7. *Onset of Respiratory Infections in Relation to Onset of Poliomyelitis; 10 Patients, by Two-Day Intervals.*

oldest, were most likely to have acquired immunity to poliomyelitis from previous contact with the virus is not a satisfactory explanation. Although all 23 of the 17- and 18-year-old boys in the sixth form had no signs of poliomyelitis, 5 (17 per cent) of the 17- and 18-year-old boys in the fifth form contracted the disease. Part of the explanation may be the relative isolation of the sixth form from the rest of the school. Members of this form had their own dormitory, sat in the back rows at chapel, ate at the head table and studied in their own rooms. On the other hand, boys living normally in a single building are not biologically isolated from their fellows, and ample opportunity exists for the contact spread of virus infections like measles and rubella among all groups in such a school. Inapparent infection as well as relative isolation and true immunity may account for the freedom from clinical disease observed in the sixth form. Inapparent infection would be consistent with the concept that upper-respiratory infections played an accessory role in determining clinical manifestations of poliomyelitis. During the month of May only one of 30 members of the sixth form was hospitalized for upper-respiratory infection.

### Origin of the Outbreak

The explosive occurrence of 5 cases within a three-day period indicated a common source of infection. The school milk supply was from the same dairy and vat as a larger quantity distributed elsewhere; no cases occurred among the other users. Source of infection in milk contaminated at the dairy was thus unlikely, but contamination at the school during distribution from eight-quart cans was not excluded. Negative evidence ruled out an ascertainable connection with the eight previously reported cases in Massachusetts. However, illness within a family residing in the town of Southboro is sufficiently suggestive to describe briefly. A patient with infectious polyneuritis (Guillain–Barré syndrome) may have had the polyneuritic form of poliomyelitis, and she or her sister, a waitress at the school, may have been the source case. The circumstance and the relationship are speculative.

Muriel J., a 16-year-old resident of Southboro, was a patient at the Massachusetts General Hospital from January 30 to February 18, 1936, with polyneuritis of undetermined origin following a head cold and a cough a few weeks earlier. Clinical findings included flaccid paralysis of the lower extremities, bilateral facial weakness and a high total protein of the spinal fluid, 105 mg. per 100 cc. By the middle of March she had recovered enough to walk without unusual fatigue. By early May she was doing everything that a normal girl might be expected to do, even swimming in the St. Mark's pool. She is certain of that fact, though not of the dates, because when the pool was drained about May 20 she went back to search for a ring she had lost. It is unlikely that she was swimming in April, for use of the pool by townspeople was restricted to evenings and temperatures then were below 40°F., except for five days. Early May, however, was the warmest since 1918, with maximum temperatures for Boston and vicinity 89 and 91°F. on May 8 and 9. During this warm spell the boys were swimming by day, and a few of the townspeople at night. The pool was formed by a dam across a natural basin, filled only in the spring and early summer by untreated ground water. One of five 10-cc. specimens collected on May 19, 1936, contained *B. coli*.

Four of the first 5 boys with poliomyelitis swam in the pool frequently prior to their infection. One, whose temperature chart is shown in Figure 6, swam daily and came directly from the pool to the infirmary with a sore throat on May 10. He developed signs of poliomyelitis on May 17. Another was in the pool on May 12 and 13 while he had a marked sore throat. Two others are known to have been swimming at some time on May 12.

The fifth boy, however, had not swum prior to his illness.

Muriel's only contact with these 5 boys was indirect, through the swimming pool and through a sister. Muriel knew none of the boys personally and did not work at the school. However, she often came to the school to visit her sister, who worked there as a waitress. On May 1, the sister developed a sore throat and remained in bed two days, returning to work on May 4.

If Muriel's illness was the polyneuritic form of poliomyelitis, it would seem reasonable to postulate that her sister also was infected on about May 1, disseminating the virus to the first 5 students as a food handler, — filling glasses with milk, for example, — around May 4 to 7. If Muriel's Guillain–Barré disease were the connecting link between poliomyelitis during the epidemic year of 1935 and the outbreak at St. Mark's School early in 1936, such an occurrence would have a precedent in the genesis of an epidemic among Eskimos described by Peart and others.[21] This outbreak, which occurred at isolated Chesterfield Inlet near the Arctic Circle on Hudson Bay in February, 1949, ultimately involved more than 50 persons with frank paralysis. Evidence indicates that a missionary with no signs of poliomyelitis probably introduced the disease into the community, where the first recognizable "sentinel case" was a patient diagnosed tentatively as having Guillain–Barré's disease and later proved to have had poliomyelitis.[22]

## Course of the Outbreak

After the first wave of 5 common-source infections developing from May 16 to 18, a second wave of 9 cases occurred between May 21 and 25, while the remaining 8 boys manifested neurologic signs of poliomyelitis between May 28 and June 1. Close contact between patients of May 16-18 and all succeeding patients can be demonstrated by adjacent seatings in study hall and classrooms or at table and by close friendships, sharing of rooms and the like. For example, the varsity baseball pitcher developed paralysis on May 17, the second baseman on May 21. One student, in the school infirmary from May 15 to May 19 with a sore throat, had ample opportunity for exposure to patients involved in the first wave of infection. This boy left school, accompanied by his brother, on May 19. Subsequently, he developed clinical signs of infection in Long Island, N. Y., on May 25; his brother developed similar signs on May 30. Likewise, 2 cousins traveled to Chicago together on May 20. One had been sitting in chapel next to the student who first manifested poliomyelitis on May 16 — by vomiting in chapel. One cousin subsequently developed paralysis on May 25, the second on May 30.

Since the significance of such evidence is only presumptive, it is not set forth in detail. Five significant facts were brought out: From about May 12 to 16, at least 5 carriers are known to have been present and distributed in every large dormitory and classroom grouping except those of the sixth form; at various periods from May 16 to 18 these five patients were hospitalized in the infirmary; of 15 boys being treated in the hospital for at least twelve hours from May 17 to 20, 5 (30 per cent) developed signs of poliomyelitis five to eleven days later; no cases occurred among the 30 boys in the sixth form who comprised 15 to 18 per cent of the school population; seven of the last 11 cases developed in boys leaving school about May 20 at subsequent intervals that varied from three to eleven days, with an average of about seven and a half days, thus establishing the minimum incubation periods for seven cases.

## Discussion

The St. Mark's School epidemic of poliomyelitis was distinctive in several respects: It occurred in the off-season of an off-year; it was sharply localized and was of explosive onset with a high attack rate; and it followed a higher incidence of spring respiratory infections than has been subsequently observed over a ten-year period. These facts suggest a concatenation of circumstances at St. Mark's School in May, 1936, that favored dissemination of the virus and development of clinically manifest disease: a grouping of susceptibles, as proved by the high attack rate, and a relatively sudden and common exposure of 5 persons who thereafter served as reservoirs of infection. Even postulating that all 190 boys were infected, the number (13) showing paresis or paralysis gives an attack rate some 10 times greater than the lifetime rate for the general population — and this within a period of two weeks.

Search has been made, therefore, for an enabling or conditioning factor that may have enhanced the susceptibility of the group. The only plausible phenomenon that involved many students was the outbreak of respiratory infections previously described. The possibility is raised that intersecting waves of 2 disease entities created circumstances favorable for the development of paralytic disease, as contrasted with simple infection. Thus, 10 boys developed signs of poliomyelitis about nine and a half days, on the average, after an upper-respiratory infection. That this was more than a chance occurrence is suggested by the high incidence of bulbar signs in this group, compared with schoolmates with no tangible evidence of clinical illness before poliomyelitis and compared with the expected frequency of bulbar disease in general populations. The clinical course of 9 of these boys was compatible with the diagnosis of what is paradoxically called the "dromedary" type of infantile paralysis.

Such an association of poliomyelitis with prevalent minor illnesses has been observed before, although its significance remains unestablished. Wilson,[23]

commenting on the experience of the Children's Hospital of Boston with some 160 to 170 patients during the epidemic period of 1935, remarked that "many had swollen cervical glands and very real evidence of tonsillitis and laryngeal infection." Others had had tonsillectomy thirteen to fifteen days prior to poliomyelitis. Five others had dysentery and poliomyelitis at the same time.

Similar observations have been recorded recently by Shaw and his colleagues[15] after an experience with 453 patients admitted to a children's hospital from 1943 to 1949: "There occurred, five days to one month prior to the onset of CNS symptoms, eight instances of acute contagious disease including rubella, epidemic parotitis, varicella, pertussis and scarlet fever; there were 4 cases of pneumonia and 7 of otitis media. There were 22 instances of preceding mild upper-respiratory infection, 9 moderately severe upper-respiratory infections and 40 cases with definite objective findings of tonsillar and pharyngeal infections such as follicular tonsillitis. There were numerous cases of gastrointestinal illness, fever, vomiting, diarrhea and abdominal pain occurring 5 to 14 days before the onset of CNS symptoms."

Pohl[24] found sore throat or tonsillitis to be "an early complaint" in 10 per cent of 1125 cases of poliomyelitis treated in Minneapolis during 1946, although, unlike Wilson, he was not impressed by signs of inflammation. However, sore throat occurred 10 times more frequently in the patients with eventual bulbar symptoms than in those with paralysis of spinal origin. Greenwood has reported that within the fortnight preceding the development of two cases of the disease at Uppingham School in England "six cases of high temperature, sore throat and catarrh occurred in the school."

Rubenstein and others[25] in the summer of 1946 studied dissemination of the virus among contacts of 5 boys who developed paralysis (4 cases of the bulbar type) three weeks after the opening of a boys' camp. They record that there were "a number of upper respiratory infections involving about 30 per cent of the camp population throughout the entire time (one month) of its operation." In a family outbreak described by Horstmann and Paul[13] two children developed symptoms of poliomyelitis four and ten days respectively after febrile sore throats; 2 other children had febrile sore throats without central-nervous-system signs; and two others had paralysis without known antecedent fever or sore throat.

TABLE 4. *Upper-Respiratory Infections and Poliomyelitis at St. Mark's School, April and May, 1936; Distribution of Cases by Form and Age.*

| FORM | I | II | III | IV | V | VI | TOTAL |
|---|---|---|---|---|---|---|---|
| Population | 18 | 29 | 40 | 38 | 35 | 30 | 190 |
| Average age | 12.8 | 13.8 | 14.9 | 15.6 | 17.0 | 17.9 | 15.5 |
| Cases poliomyelitis | 2 | 4 | 7 | 5 | 4 | 0 | 22 |
| Rate percentage | 11 | 14 | 16 | 15 | 11 | 0 | 12 |
| Cases upper-respiratory infections | 8 | 15 | 13 | 5 | 8 | 3 | 52 |
| Rate per cent | 44 | 55 | 33 | 13 | 23 | 10 | 27 |

Such minor respiratory and intestinal symptoms associated with the paralytic disease are usually interpreted as different stages of poliomyelitis rather than different (though interacting) disease entities. Against the first interpretation of the situation at St. Mark's School in 1936 is the fact that respiratory infections had been prevalent and gathering momentum in the school population for six weeks before paralytic poliomyelitis made its abrupt appearance.

At least since 1916 there has been a strong clinical impression that events in the month prior to the onset of meningeal signs — other than exposure to the virus — exert an influence on the clinical course of poliomyelitis. That they may do so was first demonstrated by clinical[6-8] and experimental[26] proof of the relation of tonsillectomy to the disease, and more recently by the observations of McCloskey and of Geffen that injections of immunizing agents influence the localization of paralysis. Such a relation to factors other than exposure is entirely consistent with the observed epidemiology of infantile paralysis, for annual frequencies have little of the rhythm or predictability characteristic of the immunizing diseases of childhood for which the factor of exposure determines the presence or absence of disease in susceptible persons. The inference has been made that development of paralysis often, if not generally, represents a clinical accident or complication of an ordinarily inconsequential infection in the same sense, for example, as mastoiditis following measles.[20] Under this interpretation the high attack rate of paralysis, as distinct from infection, observed at St. Mark's School represents an accident largely consequent to throat infections. Had there been no concurrent respiratory disease and had all other circumstances remained the same, would there have been as much infantile paralysis at St. Mark's School in 1936? The specific question cannot be answered, but the underlying hypothesis is believed probable and worthy of future epidemiologic and experimental study.

## SUMMARY AND CONCLUSIONS

The hypothesis that naturally occurring infections may influence the development of the clinical manifestations of poliomyelitis is examined against unpublished field data collected in 1936, when 22 cases of poliomyelitis occurred within a two-week period among 190 boys at St. Mark's School in Southboro, Massachusetts. The course of the outbreak is presented to show its time relation to a wave of 52 hospital admissions for respiratory infections developing in April and the first half of May, 1936 (Fig. 4). During the period of

May 1 through May 20, 41 per cent of boys who later developed poliomyelitis had upper-respiratory infections, while only 19 per cent of others in the school had such illness. The interval between onsets of the 2 types of illness in 10 boys averaged nine and a half days. Five of the 10 exhibited signs of bulbar involvement. These distributions carry a definite implication that upper-respiratory infection was one of multiple factors in the determination of clinical disease during an outbreak characterized by unusually high frequency of clinical manifestations among the total number of boys who could have been infected with the virus of poliomyelitis. The hypothesis is worthy of future epidemiologic and experimental study.

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# GASTROSCOPIC BIOPSY IN THE DIFFERENTIAL DIAGNOSIS OF GASTRITIS AND CARCINOMA*


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SINCE the introduction of the Benedict flexible operating gastroscope in 1948,[1] 203 biopsies of the stomach have been taken by this means in the Massachusetts General Hospital Endoscopy Clinic. No accidents and no complications have resulted. As already reported,[2-5] the advantages of this instrument are: biopsy of the stomach under direct vision, aspiration of gastric secretions and inflation and deflation of the stomach at will.

It seems hardly necessary to point out the importance of gastroscopic biopsy in the differentiation of benign and malignant lesions of the stomach. In all other endoscopies biopsy is practiced routinely, and its advantages are well recognized. Until 1948, however, flexible-tube gastroscopic biopsy was impossible. Aspiration of gastric secretions is now carried out by means of the operating gastroscope whenever it is desirable to clear away secretions and improve visibility or to aspirate secretions for cytologic study. In most of the following cases secretions were aspirated for cytologic examination, but since this report does not primarily concern the cytologic method the results will not be discussed. Inflation and deflation of the stomach is an important method of determining the flexibility or rigidity of the stomach wall. The presence of rigidity makes one suspicious of a neoplastic process. Repeated inflation and deflation at the will of the examiner is possible only by means of the special channel in the operating gastroscope.

The following cases, in which the clinical and x-ray diagnosis was doubtful, illustrate the value of gastroscopic biopsy.

### Carcinoma Proved by Biopsy

Case I. H. B. (PH U No. 689015), a 71-year-old man with known pernicious anemia, was referred to this hospital for gastroscopy because of epigastric pain, decreasing appetite, a large liver and equivocal x-ray reports. The pernicious anemia had been known for 3 years and was well controlled by liver therapy. X-ray films of the stomach made at another hospital a year prior to admission showed hypertrophy of the folds and possible gastric polyps. Five months before admission the same radiologist demonstrated a filling defect with multiple polyps, but there was no evidence of malignancy as shown by infiltration of the muscularis. One month before admission another gastrointestinal series was performed at the same outside hospital, at which time a small


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gastric ulcer was demonstrated without much rigidity but with progression in size of the polyps.

Gastroscopy showed, throughout the body of the stomach on the greater curvature and extending onto both anterior and posterior walls, a rounded ulcerating, sloughing, easily bleeding mass consistent with carcinoma. A satisfactory biopsy was obtained. The gross appearance was consistent with extensive carcinoma of the stomach; positive proof was obtained by the biopsy, which showed adenocarcinoma.

*Comment.* Gastroscopic biopsy in this case established a positive diagnosis of carcinoma of the stomach, whereas x-ray examination had been doubtful.

CASE 2. J. P. (MGH U No. 182159), a 63-year-old man with known pernicious anemia of 13 years' duration, was referred for gastroscopy because of weakness, fatigue, recent failure to respond to liver therapy and inconclusive x-ray studies. Four gastrointestinal series carried out in the preceding 5 months had resulted in varied and doubtful diagnoses, the most recent examination having been reported as essentially normal.

Gastroscopy demonstrated a lesion approximately 4 cm. in diameter lying on the anterior wall in the upper part of the body of the stomach. An ulceration 1 cm. deep was observed in the center of it. The edges were elevated above the surrounding mucosa by about 3 or 4 mm. The whole lesion was definitely circumscribed and was thought to be a carcinoma of low-grade malignancy. A biopsy was obtained from the central portion. The pathological report was adenocarcinoma.

*Comment.* After four equivocal x-ray examinations, gastroscopy demonstrated a definite proliferating lesion of the stomach, which was proved by gastroscopic biopsy to be adenocarcinoma.

CASE 3. C. F. (BM U No. 727806), a 27-year-old man, entered the hospital with a diagnosis of gastric ulcer. His chief complaint was increasing abdominal distress of 4 months' duration. A gastrointestinal series made in another city was said to show a preantral gastric ulcer with severe hypertrophic gastritis and giant rugae. Dietary treatment had not helped the patient. At physical examination on admission he was found to be extremely agitated; on the next day he was writhing in bed, cursing loudly and complaining bitterly of epigastric pain. His bizarre behavior made it necessary to consult a psychiatrist. X-ray examination disclosed marked thickening of the gastric folds, with questionable areas of ulceration along the lesser curvature. There was a conspicuous 3-cm. defect in the antrum, probably resulting from greatly thickened folds. Severe gastritis was thought probable; however, neoplasm, such as diffuse lymphoma of the stomach, was not excluded.

Gastroscopy was performed twice. The first time it was unsatisfactory because of lack of co-operation on the part of the patient. On the second occasion the stomach contained a large amount of coffee-ground material, which was aspirated in order to improve the visibility. The rugae on the greater curvature showed increased reddening, and in some places there was a segmented appearance (high up in the body of the stomach). Lower down in the body, near the angulus, there was a very ragged appearance, with increased reddening and edema. The angulus was difficult to identify because of the patient's constant belching, which made the examiner suspicious of some rigidity. No peristalsis was visible. The findings were consistent with severe gastritis. It was thought that lymphoma should be seriously considered. Three biopsies were obtained from the greater curvature of the midportion of the body of the stomach. The pathological report was carcinoma.

*Comment.* In this young man, only 27 years of age and apparently with a psychopathic personality, it was hard to believe that he had anything more than a severe gastritis. Gastroscopic biopsy, however, proved a diagnosis of colloid carcinoma.

CASE 4. O. B. (MGH U No. 695057), a 70-year-old man, was referred for gastroscopy because of unexplained gastrointestinal bleeding, 17 years after excision of a benign gastric ulcer, pylorectomy and posterior gastroenterostomy. X-ray examination of the stomach showed widened gastric and jejunal folds.

Gastroscopy was performed 2 days after the x-ray study and revealed 4 or 5 erosions surrounding a somewhat irregular ulceration 1½ cm. in diameter that had a gray base but somewhat irregular margins. Its depth was estimated at 2 or 3 cm. A biopsy was taken from the mucosa of the lesser curvature near the angulus but not directly from the ulcer. The findings were those of a 1½-cm. gastric ulcer and multiple erosions. There was some rigidity in the area of the ulcer and a definite suspicion of malignancy. The pathological report was adenocarcinoma.

*Comment.* In this case gastroscopy with biopsy was the only means of making a positive diagnosis. X-ray films showed only gastritis. The gross appearance at gastroscopy was not positive for carcinoma, but the biopsy obtained through the gastroscope was unequivocally diagnostic of adenocarcinoma.

CASE 5. J. S. (BM U No. 671259), a 64-year-old man, was referred for gastroscopy because of hypogastric pain, loss of weight and doubtful x-ray examination. Gastrointestinal series before admission showed a gastric ulcer with irregularity of the lesser-curvature aspect of the stomach, some of which was due to large rugae. Malignancy could not be excluded.

Gastroscopy demonstrated an irregularity on the lesser curvature of the angulus extending up almost to the cardia. This was ulcerating, bleeding and nodular. It seemed to extend well onto the posterior wall, appeared rigid and did not respond to air inflation. The findings were thought to be consistent with an extensive nodular, ulcerating carcinoma. A satisfactory biopsy was obtained, which was reported as colloid carcinoma.

*Comment.* In this case gastroscopic observation with biopsy proved a diagnosis of colloid carcinoma.

## *Discussion*

It is interesting to note that pernicious anemia was present in Cases 1 and 2. Gastric atrophy is usually present in untreated pernicious anemia and is probably a predisposing factor in the development of gastric carcinoma.[6, 7] All patients with pernicious anemia should therefore be examined frequently by x-ray as an aid in the diagnosis of possible early carcinoma. X-ray examination should be supplemented at once by gastroscopy when there is any doubt in the mind of the clinician or radiologist. In Cases 1 and 2 the clinical findings were highly suggestive of carcinoma of the stomach, but the x-ray films were inconclusive.

Case 3 is unique because it concerned an apparently psychoneurotic young man only 27 years old in whom a diagnosis of carcinoma of the stomach seemed at first unlikely. The radiologists suspected severe gastritis or possibly lymphoma.

In Case 4 there had been an excision of a benign gastric ulcer with pylorectomy and gastroenterostomy 17 years previously. X-ray examination of such a distorted stomach is always difficult, and the radiologist was quick to realize his handicaps and to request help from the gastroscopist. Here again, as in previous articles, I wish to re-emphasize the fact that the two methods are complementary and should be used to supplement each other. The limitations of the radiologist are not the same as the limitations of the gastroscopist. A blind area by one method may be an easily visible area by the other method.[8, 9]

In Case 5 both clinician and radiologist suspected gastric carcinoma, but it could be proved only by gastroscopic biopsy.

## Normal Mucosa or Gastritis Proved by Biopsy

Case 6. B. L. (MGH U No. 127907), a 66-year-old man, was referred for gastroscopy because of weakness, 25-pound weight loss, tarry stools and an x-ray examination reported as showing huge folds in the fundus of the stomach and along the greater curvature, suggesting gastritis or possible lymphoma.

Gastroscopy disclosed edematous rugae along the greater curvature, flexible and not verrucous. Higher up in the fundus of the stomach the rugae became a little roughened and irregular but still were flexible. They bled easily; no erosion, ulceration or neoplasm was demonstrable. A biopsy was taken from one of the large folds of the greater curvature, in the midportion of the body of the stomach. The gross findings suggested some degree of superficial gastritis; lymphoma seemed unlikely. The pathological report was "no diagnostic abnormality recognized."

Seven months later x-ray examination of the stomach was entirely negative.

*Comment.* In this case a biopsy of the stomach was important in almost certainly excluding such a diffuse process as lymphoma. It also tends to show that thick edematous folds as seen by x-ray and by gastroscopy may not signify gastritis.

Case 7. I. K. B. (PH U No. 704169), a 62-year-old woman, was referred for gastroscopy because of the possibility of recurrent carcinoma of the stomach following subtotal gastrectomy 10 months previously. Since the operation she had suffered from anorexia, loss of weight and a sense of fullness after eating. She frequently regurgitated small amounts of food. X-ray examination of the gastric remnant revealed no intrinsic filling defect or ulceration.

Gastroscopy demonstrated a normal stoma with smooth margins, slight reddening and edema on the gastric side and no evidence of recurrent carcinoma. A biopsy specimen taken from close to the edge of the stoma was reported as showing normal small-bowel mucosa, probably jejunal.

*Comment.* Gastroscopy with biopsy in this case confirmed the x-ray and gross gastroscopic impression of no intrinsic disease of the stomach. It is of special interest to note that it was possible to obtain a biopsy from the jejunal side of the stoma.

Case 8. M. M. (MGH U No. 708638), a 74-year-old woman, was referred here for gastroscopy from another hospital because of gnawing epigastric pain, relieved by Gelusil but not well relieved by food. She had lost 30 pounds in weight during the last few months and at the time of admission weighed only 57 pounds. X-ray examination at another hospital was reported as showing a constant deformity on the posterior wall of the stomach. It was in the nature of a crater and apparently represented an ulcer. No infiltration or destruction was seen around it.

Gastroscopy showed some active bleeding in the stomach, which prevented a wholly satisfactory view. Bloody secretions were aspirated in order to improve the visibility. Normal rugae were not seen on the greater curvature, but high up in that area was noted a smooth, rounded, overhanging mass that suggested tumor formation. The patient could not retain the air in the stomach; this, in addition to the bleeding, considerably hampered the examination. There seemed to be an ulceration in the smooth area, as well as another ulceration on the posterior wall. A biopsy was taken from the greater curvature high up in the body of the stomach in the smooth area. The gross findings suggested tumor formation. The pathological report, however, showed only slight chronic gastritis and no evidence of malignancy.

After gastroscopy the patient gained weight, became asymptomatic and had no occult blood in her stools. Two weeks later she returned to the same outside hospital, where a laparotomy was performed. No induration or mass could be palpated anywhere in the stomach. The stomach was opened through a longitudinal incision in the anterior wall. Palpation of the mucosa revealed chronic gastritis but no ulceration or induration. After the operation the patient's course was uneventful, and she progressed better than many patients of her age.

*Comment.* This is another case in which much of the evidence indicated carcinoma of the stomach but biopsy showed only chronic gastritis, later confirmed by surgical exploration with gastrotomy.

Case 9. S. M. (BM U No. 691546), a 62-year-old man, was referred for gastroscopy because of anorexia and loss of 18 pounds in 3 months. Many x-ray examinations of the stomach had been made prior to admission and had been reported as probable carcinoma of the stomach.

Gastroscopy demonstrated throughout the body of the stomach on the lesser curvature, anterior wall and posterior wall, extending from the angulus to the cardia, an indurated, rigid, thickened, irregular appearance with numerous areas of ulceration and depression. The normal rugal pattern was absent. There was a large amount of adherent secretion. The gastric wall did not respond well to air inflation. About 4 ounces of retained secretion was aspirated. A satisfactory biopsy was obtained from the lesser curvature in the midportion of the body of the stomach. The gross appearance was definitely suggestive of an infiltrating type of carcinoma with some rather superficial ulcerations. The pathological report, however, showed only acute and chronic gastritis.

Since the patient's appetite was inproving and he was gaining weight and strength, he continued on a medical regimen.

*Second admission.* One month later the patient was readmitted for another gastroscopy. In the interim he had been feeling very well. Gastroscopy on this occasion showed the mucosa to be still abnormal, with an irregular, beefy-red, indurated appearance, many erosions and some ulcerations up to at least 1 cm. in diameter and at least 2 or 3 mm. in depth. The beefy-red appearance alternated with white patchy areas, and the stomach did not respond as well as usual to air inflation. No normal rugae were present. The process seemed to involve both curvatures and both anterior and posterior walls. Two biopsies were taken. The gastroscopist believed that diffuse gastritis might give this appearance but it should have shown more improvement in the last month, especially in view of the patient's 12-pound weight gain and general feeling of well-being. X-ray study made by one radiologist at this time showed that the irregular filling defects of the lesser-curvature side of the stomach wall did not appear appreciably changed in extent. Malignancy was still considered likely. A second radiologist found multiple tiny ulcerations along the lesser curvature and favored a diagnosis of hypertrophic gastritis with multiple ulcerations or possible lymphoma. Gastroscopic biopsy, however, was reported as showing an extremely severe (Grade IV) chronic gastritis. The pathologist noted that the number of plasma cells in the infiltrate was unusually large and the severity of the gastritis was far beyond physiologic limits.

*Third admission.* Two months later the patient was referred for gastroscopic check-up. He had shown steady improvement since the previous admission. Gastroscopy at this time revealed a beefy-red appearance with multiple erosions, more induration than before and more elevated irregularities, with a suggestion of even greater rigidity. Biopsy, however, was reported as chronic gastritis, with no evidence of malignancy.

*Fourth admission.* Two and a half months later the patient was feeling in excellent health, was asymptomatic, had a good appetite and was "working like a horse." Gastroscopy at this time demonstrated marked improvement in the mucosa of the body of the stomach, much less induration and no evidence of rigidity. On the anterior wall in the upper part of the body of the stomach some reddening and a suggestion of a beefy appearance were still seen, with a few areas that resembled healing erosions. A good biopsy was again obtained and was reported as acute and chronic gastritis with no evidence of malignancy.

In view of the patient's continued improvement no further gastroscopies were done. A follow-up letter from this patient received 5 months after the fourth gastroscopy showed that he was feeling fine, with a good appetite, and was apparently back to normal condition. He was still careful about his diet, but he reported that things tasted good once again.

*Comment.* In this case both the x-ray findings and the gross appearance at gastroscopy suggested gastric malignancy. Repeated biopsies, however, on 4 occasions over a period of 5 months seemed adequately to rule out the possibility of neoplasm.

Case 10. L. L. (PH U No. 700372), a 54-year-old man, was referred here for gastroscopy because of heartburn, tarry stools, loss of weight and an x-ray examination suggestive of possible early neoplasm. A radiologist in another city had demonstrated a small gastric ulcer on the greater curvature

of the antrum, 2 other areas suggestive of small ulcerations, thickened rugae and minimal stiffening of the gastric wall. He was unable to distinguish between inflammatory and neoplastic infiltration.

Gastroscopy revealed a considerable amount of fluid in the stomach, which was aspirated for better visualization. Because of spasm the angulus was not seen at first but was finally found and was best seen with the indicator in the 9-to-10-o'clock position. It was considerably reddened but appeared smooth and round. The proximal antrum was visualized; its greater curvature was markedly red and edematous. No erosion, ulceration or neoplasm was demonstrable in the proximal antrum. The distal antrum and pylorus were not visible because of lack of peristalsis. On the greater curvature of the body of the stomach were seen a few eroded reddened areas, with some cobblestone appearance. A biopsy was taken from the greater curvature of the stomach near the angulus. The findings were consistent with a moderately severe gastritis. The pathological report was severe chronic gastritis.

*Comment.* In this case the radiologic appearance suggested the possibility of neoplastic infiltration. A diffuse neoplastic infiltration had probably been excluded by the gross gastroscopic appearance and by the pathological report of severe chronic gastritis. A localized carcinoma cannot be entirely ruled out by a negative biopsy.

### *Discussion*

Case 6 is interesting because the gastric rugae looked large both by x-ray and by gastroscopy but the gastroscopic biopsy showed no diagnostic abnormality. Thick folds may not mean gastritis.

In Case 7 recurrent carcinoma of the stomach was suspected but was reasonably well excluded by a negative x-ray film, negative gastroscopy and a pathological report of normal jejunal mucosa from the gastroscopic biopsy.

Cases 8, 9 and 10 may be discussed together, as gastroscopic biopsy in all three showed gastritis of some degree, slight in Case 8 and severe in the other two. In these three cases the question of malignancy was much in the foreground but was reasonably well excluded by gastroscopic biopsy and the patient's subsequent clinical course. One should be cautious, however, about putting too much faith in a biopsy showing a nonmalignant process, as the value of any endoscopic biopsy depends entirely on the endoscopist's ability to secure a proper tissue sample. Under certain conditions any endoscopist may fail in this respect. A positive biopsy proving malignancy is therefore more valuable than a negative biopsy, which can only exclude malignancy for a particular sample. Nevertheless, when a *diffuse* carcinoma or lymphoma is suspected, considerable reliance may be placed on a gastroscopic biopsy that does not show it.

## Conclusions

Biopsy of the stomach as obtained through the flexible operating gastroscope is of great value in the differential diagnosis of malignant tumor and gastritis.

Two hundred and three satisfactory gastroscopic biopsies have been obtained without any accidents or complications.

Ten illustrative cases are reported herewith. In 5 cases a positive diagnosis of carcinoma was established by gastroscopic biopsy. In the other 5 diffuse gastric malignancy was excluded with reasonable certainty by gastroscopic examination and biopsy.

It is reasonable to state that no gastroscopic examination is complete unless biopsy under direct vision is available.

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# FULL-TERM ABDOMINAL PREGNANCY

## A Case Report

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ALTHOUGH ectopic pregnancy is reported as occurring once in 100 pregnancies, only 1 per cent of extrauterine pregnancies reach the stage of viability, — an incidence of approximately 1:10,000. The incidence of abdominal pregnancy is 16 times higher in Negresses than among white women, is higher in women over the age of 30 and is more often seen in a first or second pregnancy. In 1935 Hellman and Simon[1] calculated that 316 authentic instances of abdominal pregnancy had been reported up to that time. Mitra[2] of India has stated that he had found 420 cases up to 1938 in the world literature; in his own experience advanced abdominal pregnancy had occurred twice successively in the same patient. In April, 1948, Ware[3] added 249 cases of seven months' duration or longer to those previously reported by Hellman and Simon, to bring the total to 565.

The fetal loss and the high incidence of congenital anomalies (whether due to ovum defects or to subsequent developmental or pressure factors) are noteworthy in abdominal pregnancies. The fetal loss runs as high as 76.5 per cent in extrauterine pregnancies of more than five months' gestation; if a living, viable baby is obtained, his chances of survival for eight days or more are rated as only 44.3 to 57.1 per cent.[4] The percentage of reported deformities, moreover, ranges from 10 to 46 per cent. The maternal mortality in recent years has been reduced to about fifteen per cent from a previous 35 per cent,[12] although Beacham and Beacham[5] reported only one death in 20 cases.

Abdominal pregnancies are considered either primary or secondary. Doubt has been raised whether implantation of the ovum ever really occurs primarily in the abdomen; however, in very rare instances this may possibly take place, and apparently authentic cases have been reported. Jarcho[6] suggests that such ova may be implanted and develop on ectopic endometrium in the abdominal cavity. Litzenberg[7] lists the commonly accepted criteria for determining a primary abdominal pregnancy: normal tubes, ovaries and broad ligaments, except at the site of implantation; absence of intraligamentary rupture of a tube; and lack of evidence of escape of the ovum from the uterine cavity.

Abdominal pregnancies have been further classified by Schumann[8] under the following headings: Extraperitoneal, intraligamentary implantation — in this type the fetus develops between the leaves of the broad ligament; tuboabdominal pregnancy — the implant remains attached to the wall of the tube after rupture (both these types are quite rare); secondary abdominal pregnancy — this form of abdominal implantation is perhaps the commonest, occurring after tubal abortion or after tubal rupture. The ovum may become attached anywhere in the lower abdomen; the placenta develops a new blood supply; tubo-ovarian pregnancy —in which the implantation and development of the placenta takes place on the tube and ovary; and ovarian pregnancy — another rare type, as a primary implantation.

Other rare extrauterine pregnancies, such as cervical pregnancy or pregnancy in a rudimentary horn, do not reach the stage of viability.

Most early pregnancies that are discharged into the abdominal cavity, — if not removed at operation, — will either absorb or become sealed off by adhesions, with the fetus showing signs of maceration, or of adipocere or lithopedion formation. The literature contains numerous instances of old abdominal pregnancy found years afterwards when calcification had taken place. It is a lucky fetus whose placenta happens to become attached to an organ whose blood supply permits growth and development to take place and where the environment allows the baby to be delivered at operation as a normal, healthy infant.

The following case report is "a case of the rarest type of abdominal pregnancy, — a full term surviving infant without congenital anomalies or deformities and a surviving mother."[9]

### CASE REPORT

M. B., a 39-year-old para ii, gravida iii, was first seen on October 25, 1946, with a history of bloody vaginal discharge of 10 days' duration following a 2-month period of amenorrhea. She had also had a steady, bearing-down pain in the lower abdomen for a week, with severe low backache, radiating down the right thigh. The vaginal flow had required the use of only one pad a day, with passage of a few small clots. The breasts were rather sore, and there was some frequency.

Menstrual periods had begun at age 14 but were quite irregular, with intervals varying from 2 to 8 weeks. During the past 5 years the menstrual flow had been more profuse, now lasting up to 7 days, with 18 or more pads used at a time and with passage of clots. The patient had felt hot flashes for the past 4 years. She had had two previous pregnancies: the first, in 1931, when she had a normal delivery of an 8¾-pound girl after 18 hours of labor, the second in 1937, when she had a normal delivery of a 7-pound girl. She had "not avoided" pregnancy in recent years. Past history included no accidents or previous operations and no serious

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illnesses except diphtheria at the age of 5. She had been treated recently by her family physician for low blood pressure and anemia and had had her cervix cauterized because of two cysts. The family history was negative except that the patient's mother had died of cerebral hemorrhage. She had been married for 17 years; her husband was living and well. Her two daughters, aged 15 and 9, were living and well.

Physical examination showed a small woman (115 pounds) in no acute pain and not appearing acutely ill. The blood pressure was 130/78. Physical examination was negative except that the breasts were soft, with no masses, but with prominent Montgomery tubercles; the abdomen showed no scars, masses, spasm or tenderness; muscle tone was fair. Vaginal examination showed a parous outlet, with a slight rectocele, and with a stringy, dark, bloody discharge coming from the cervical canal and a negative Chadwick sign. The cervix was patulous but only slightly softened, with no erosion or cervicitis; the uterus was retrocessed, quite firm in consistency, freely movable and apparently enlarged to the size of a 6- to 8-week pregnancy, but with some irregularity on the anterior surface; there was no tenderness or masses of the adnexa.

The impression after examination was that of a fibroid uterus, with an early pregnancy superimposed, — either incomplete abortion or possible ectopic pregnancy. Hospitalization was advised for a pregnancy test and possible operation, but this was refused; the patient was finally discharged to her home, in another town, to remain in bed and to report in 3 days. At the end of this time she reported by letter that there was no more pain, that bleeding had stopped and that she was feeling well; re-examination in another month was advised.

At her second visit, on December 7, 1946, the patient stated that there had been no more vaginal bleeding since the first examination. However, there had been severe lower-abdominal pain, worst during the preceding week; her family physician had been called in and had given her a hypodermic for relief. There had been considerble nausea, without vomiting, and some yellow vaginal discharge. Her weight was 112 pounds; examination of the urine was negative; the blood pressure was 134/80. There was a palpable mass in the right lower quadrant of the abdomen, and the fundus measured 8 cm. above the symphysis. Vaginal examination showed a 2½-month pregnancy, with the uterus apparently in 2° retroversion and with a tender "subserous fibroid," the size of a golf ball, on the right anterior wall.

During the next few weeks the abdominal pain persisted, with little relief from aspirin and codeine, and again morphine was prescribed by the patient's family physician. She first began to feel life toward the end of December. A blood Hinton test was negative, the hemoglobin was 85 per cent (Tallqvist) and the blood was found to be Group O, Rh positive. The patient was started on calcium and iron by mouth, and was seen at 2- to 3-week intervals. The lower-abdominal pain gradually decreased, being replaced by considerable discomfort in the left flank and left upper quadrant, with considerable distention of the abdomen. The height of the fundus seemed to increase steadily, but the head of the fetus became readily palpable in the region of the left flank. Weight gain was not excessive (14 pounds), the urine was negative for albumin and sugar and the blood pressure was within normal limits. During the last 2 months 3 attempts were made to perform an external version of the fetus, but since the fetus did not turn readily attempts were abandoned. Vaginal examination on April 26, 1947, showed the presence of small parts in the pelvis, but no further abnormality was recognized at that time. Pelvic measurements were adequate, the smallest measurement being the diagonal conjugate (12.5 cm). On May 24, 1947, at the last office visit, the fundus measured 22 cm. above the symphysis, the blood pressure was 144/86, the urine was negative and the patient's weight was 126½ pounds. At this visit the patient complained of almost constant abdominal discomfort, especially in the left flank and lower abdomen. Pain was excessive when the fetus moved, but no regularity of pains was discernible, and she was considered not to be in true labor. There was no bloody show, but because of excessive pain the patient was admitted to St. Luke's Hospital on May 26, 1947, during the night.

Physical examination at this time showed a fairly well developed and nourished woman, apparently in considerable discomfort. The temperature was 97.8°, the pulse 60 and the blood pressure 128/86. Abdominal palpation suggested a fundus lower than expected at term, although the fetus appeared to be that of an 8½-month pregnancy. The head of the fetus was felt in the left flank, but abdominal palpation was difficult because of considerable muscular rigidity. The fetal heart sounds were regular. No recurrent contractions were made out. Rectal examination showed the presenting part to be high, with small parts palpable at the pelvic brim. The cervix was neither effaced nor dilated. No other pelvic masses were made out. The impression was of fibroid uterus, near term, with transverse-lie presentation.

During the next 36 hours the picture changed little, with no regular contractions noted, but with the patient crying out frequently because of abdominal pain, mostly below the umbilicus and in the right lower quadrant. There was no further dilatation of the cervix. The patient took a liquid diet and did not vomit. Sedation with Nembutal only was used at intervals of several hours. Laboratory studies showed a negative urine, with 4,160,000 red blood cells and a hemoglobin of 12.6 gm., and 12,000 white blood cells with 75 per cent neutrophils and 25 per cent lymphocytes. No vaginal examination was done at term. An x-ray film of the abdomen was taken and was reported as showing "a fetus in utero with the breech presenting." Re-examination of the x-ray film at a later date failed to show a separate uterine shadow. Another obstetrician was called in consultation, but again abdominal palpation was difficult, causing the patient much pain. Cesarean section was decided on in view of the age of patient, the transverse presentation and the fact that the patient was not in labor and was intensely uncomfortable.

At laparotomy, performed under spinal anesthesia on May 27, 1947, numerous adhesions were encountered between a large membranous sac and the parietal peritoneum and the internal organs. These adhesions separated easily and the fetal sac was then opened, with a small amount of gray amniotic fluid escaping. The baby was a girl, weighing 5 pounds, 6 ounces, and lay transversely with the vertex in the left flank. Delivery was accomplished at 5:39 p.m., and the baby cried at once after ligation of the cord. The cord had a membranous insertion and there were numerous areas of dark blood in the placenta beneath the amnion. The placenta was found to be densely adherent to the fundus of the uterus in the region of the right cornu. The right tube appeared to have been severed, as the distal 12 cm. was attached to the placenta, receiving its blood supply from the broad ligament, but without any direct connection with the uterus. The right ovary appeared normal. The uterus was about the size of a 2½-month pregnancy, being symmetrical, firm and with no evidence of fibroid tumor. The left tube appeared normal. The left ovary was not clearly visualized, being buried under a mass of adhesions behind the left broad ligament. Hysterectomy was decided on after due consideration of the patient's age, her parity and the firm attachment of the placenta to the uterus. A supravaginal hysterectomy with right salpingectomy was then quickly performed and the abdomen closed without drainage. Five gm. of sulfanilamide crystals was sprinkled in the cul-de-sac. One unit of plasma and 500 cc. of glucose were given intravenously at operation; blood transfusion was not considered necessary.

The patient's postoperative course was smooth and afebrile, with no temperature elevation over 99°. No antibiotics were given. Prostigmine was used for the abdominal distention that occurred, and intravenous fluids were given to counteract the postoperative nausea and vomiting during the first day. Bathroom privileges were granted early; when the clips were removed on the 7th day the incision was found to be healing well. Examination of the baby showed her to be apparently normal. She nursed well and gained rapidly, weighing 6 pounds on breast milk alone when discharged with the mother on June 10, 1947.

At the 6 weeks' check-up examination on July 19 the baby was still nursing and weighed 8 pounds, 12 ounces. The mother was in good condition, also, complaining only of some epigastric discomfort and occasional gas pains; her bowel movements were normal. The operative wound had healed, and the patient was treated for a mild trichomonas infection of the vagina, which responded promptly to therapy. She was last seen in the office in May, 1948, about 11 months after the operation, complaining of headaches and a moderate rectocele; the baby was still thriving and weighed 21½ pounds.

The pathological report reads as follows: "Specimen consists of a uterus amputated above the cervix with a large placenta of a full term pregnancy attached to its upper, outer surface (Fig. 1). The uterus is only slightly enlarged and

measures approximately 10 x 8 x 5.5 cm. For the most part, the serosal surface is smooth and glistening. There is a short piece of the left fallopian tube attached. Examination shows that the placental attachment is not only along the superior border of the uterus but it also extends down the right lateral wall, displacing the right fallopian tube far out on the edge of the placenta (Fig. 2). The peritoneum is reflected from the upper surface of the uterus to cover the entire placenta. It is this layer apparently that has helped to keep the extrauterine pregnancy from perforation. The fetal membranes, cotyledons and a considerable portion (30 cm.) of the umbilical cord are included in the placenta. The cord is not attached over the cotyledons but out along the membranes 8 cm. from the periphery of the cotyledons. From this point, greatly distended blood vessels extend out into the fetal membranes and cotyledons. Numerous hemorrhages are seen in the placenta. The displaced right fallopian tube is

FIGURE 1. *Anterior Aspect of Uterus and Placenta, Showing the Numerous Blood Vessels that Supplied the Pregnancy and the Widespread Areas of Hemorrhage in the Placenta.*

stretched along the periphery of the placenta and measures 16 cm. in length. On section the myometrium is found to be moderately firm and pale and measures 4 cm. in thickness. The endometrium is thick and somewhat congested. A probe inserted into the right cornu passes out into the placenta. Further section at this point shows that in this region the placenta has split the myometrium and it extends almost to the endometrium in the fundus. It appears that the pregnancy developed first in the tubal insertion in the right cornu of the uterus. Microscopic diagnosis: Extrauterine pregnancy (full term) with secondary abdominal development."

## DISCUSSION

Probably because of the rarity of the condition, abdominal pregnancy is often not diagnosed in time to save the baby. Less than half the cases are diagnosed preoperatively,[2, 10] and in this case also an incorrect diagnosis was made of an abnormal intrauterine presentation complicated by fibroid. The history of bleeding and pain early in pregnancy, at which time ectopic pregnancy was suspected, is of course rather typical of most cases of abdominal pregnancy. The lack of tenderness at the initial vaginal examination, the subsequent amelioration of symptoms and the steady growth of the fetus all indicated an intrauterine pregnancy.

Toward the end of the patient's pregnancy the marked abdominal discomfort, with nausea, and the lack of regularity in the recurrent pain were typical. There were no palpable uterine contractions, and discomfort was most marked when the fetus moved or when the abdomen was palpated. Although small parts are claimed to be readily palpable in such cases, similar findings are noted in multiparas with thin abdomens, whereas in this patient palpation was difficult because of muscular spasm. A high transverse or diagonal presentation should, of course, make one pause to consider the cause of malpresentation; external version, as in this case, is not always the answer to such a problem. Because of the high station of the presenting parts, little can be gained by vaginal examination, although a firm cervix high in the vaginal vault and a uterus separate from the fetal mass are described

FIGURE 2. *Uterus with Attached Placenta Seen from the Posterior Aspect, Showing the Membranous Insertion of the Cord, and the Visceral Peritoneum Covering the Placenta, which is Firmly Adherent at the Right Cornu.*

*A probe has been inserted into the displaced distal portion of the right fallopian tube.*

as helpful findings. X-ray examination may be of help in confirming a diagnosis already suspected, but it was of no help in this case.

The preferred time of operation, when the diagnosis of abdominal pregnancy is once made, is usually the thirty-sixth to the thirty-eighth week, — at a time when the fetus is large enough to be viable but before the period at term when intra-abdominal death frequently occurs. If the fetus is already believed to be dead it is considered wise to allow a period of several weeks or months to elapse before operation, to facilitate the separation of the placenta.

There has been some controversy over the treatment of the placenta. In general, it is preferable to remove the placenta in toto if such removal can

be readily accomplished without danger to the patient; otherwise it is better left in place. All writers on this condition agree that removal of only a part of the placenta will cause excessive bleeding, with marked increase in the maternal mortality.[2, 9-11] Marsupialization of the placenta or packing and drainage, formerly used when it was firmly adherent, are now employed only for cases that may show frank infection. On the other hand, leaving the placenta attached (as advised by Beck[12]) and closing the abdomen has become an accepted and advisable procedure in many instances. The placenta often becomes absorbed without residual adhesions, although liquefaction and suppuration also may occur, requiring subsequent operation.

Concomitantly with a more intelligent approach to the handling of the placenta the recent advances in surgery, with blood transfusion, plasma and coagulants to combat the problem of hemorrhage, have undoubtedly helped to lower the maternal mortality in recent years. Added to these are the various antibiotics and chemotherapeutic agents that have helped to reduce infection and thereby reduce morbidity and shorten convalescence.

It is interesting to speculate, from the history and from the evidence found at operation, how adequate circulation was provided to support this pregnancy through its development. One might conjecture that when the patient was first seen, with ten days of spotting and pelvic pain but with no localized tenderness, the tubal pregnancy, which had established itself in the interstitial portion at the right cornu, had not yet broken through to the serosal surface. During the week before the second visit, when the patient required a hypodermic from her family physician, the partial rupture presumably occurred, without evidence of shock or of internal bleeding. Thereafter there must have been a gradual extrusion of the amniotic sac through the gap in the muscle, but without complete detachment of the sac from its blood supply. According to Schumann's classification, therefore, this is a case of tuboabdominal pregnancy. In the process of growth the occluded right fallopian tube was elongated and the visceral peritoneum stretched out to cover the placenta and membranes, thereby giving added protection to the fetus. A similar case has been recently described by Waters.[13]

The exact duration of this pregnancy is uncertain, because of the patient's history of irregular menstruation (two- to eight-week interval), but with allowance for this factor the pregnancy is estimated as of thirty-eight to forty weeks' duration. The placenta, as is often the case, showed numerous areas of hemorrhage, with dark blood infiltrating beneath the amniotic layer. This placental hemorrhage probably accounts for the high incidence of fetal death near term in abdominal pregnancy, especially when the fetus may already be stunted by an inadequate blood supply.

## Summary

A 39-year-old multipara was operated on for abdominal pregnancy near term, with delivery of a normal living female infant weighing 5 pounds, 6 ounces and with a smooth postoperative course.

The author wishes to express his thanks to Dr. Theodore W. Jones for his assistance on this case; to Dr. Eric F. Joslin for the pathological study; and to Dr. Garvey Adeson for the photographs of the placenta.

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# DECLINE IN MORTALITY IN ACUTE RHEUMATIC FEVER IN NEW HAVEN, CONNECTICUT, 1920–1948*

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NEW HAVEN, CONNECTICUT

IT HAS been suspected for two decades or more, both in the United States and in England, that the prevalence of rheumatic fever is declining. It has not been easy to document the fact in this country, as this disease and rheumatic heart disease§ are not reportable here. However, some reliance can be placed on morbidity and mortality surveys, and by means of such methods, crude as they are, evidence is now beginning to accumulate that the decline in morbidity rates for rheumatic fever and mortality rates for rheumatic heart disease is an actuality. Studies confirming this impression began with that of Atwater[1] in 1927, which indicated a striking fall in the number of deaths and probable cases during the previous quarter-century. In 1930 Glover,[2] in England, ventured to call acute rheumatism an obsolescent disease; sixteen years later, in 1946,[3] he presented data indicating a great decrease in its mortality in England and a change in age incidence in regard to its mortality. His data covered a period of sixty-four years, 1881 to 1944. In the United States deaths from rheumatic fever have been definitely on the decline for some thirty years, and the observations of Dublin and his co-workers,[4] Hedley[5] and Swift and Cohn[6] furnish evidence tending to confirm this belief. A select group of white children of Metropolitan Life Insurance policyholders 1 to 14 years of age showed a decline of 4 per cent in deaths due to acute rheumatic fever from 1911-1915 to 1941–1945.[7] A statistical epidemiologic investigation of childhood mortality in age groups from 5 through 19 years for three-year periods, 1919–1921, 1929–1931 and 1939–1941, supplemented by data for the single years 1942, 1943 and 1944 in the United States, has been reported recently by Wolff.[8] His findings indicated a distinct decrease in mortality from acute rheumatic fever and heart disease (90 per cent of which was probably rheumatic heart disease) for white children, but for non-white children no consistent downward trend was evident except for the age groups of 15 to 19 years; even this decrease was far behind that for the comparable white group. A recent report of Haenszel[9] has indicated that in the state of Connecticut there has been a steady decline in deaths due to acute rheumatic fever since 1905 and for all forms of heart disease 5–14 years since 1922. The fact that nearly all deaths due to heart disease in the 5-to-14 or 5-to-24-year age groups are due to rheumatic heart disease has been used as a method of obtaining the minimal mortality due to rheumatic heart disease. This procedure was used by Hedley[5] in particular, in a study of mortality due to heart disease in persons 5 to 24 years of age in the United States from 1922 to 1936.

A considerable body of knowledge concerning rheumatic fever and rheumatic heart disease in New Haven, Connecticut, has been accumulated, but the report by Deutsch[10] that covered mortality due to rheumatic fever in New Haven for the period 1935–1939 did not attempt to analyze mortality trends. It is the purpose of this paper to present data showing mortality trends for rheumatic fever and rheumatic heart disease for the city of New Haven during the period 1920–1948.

## *Methods*

The death certificates covering the years 1920–1948, inclusive, on persons listed as residents of New Haven were first reviewed.

The causes of death recorded on each death certificate were carefully examined, and those bearing data that indicated the cause of death as rheumatic fever or rheumatic heart disease or one of its complications were placed in the following categories for purposes of classification:

Rheumatic heart disease plus heart failure at 25 years of age and over; acute rheumatic heart disease plus heart failure occurring in the age period of 2 to 25 years; rheumatic heart disease plus bacterial endocarditis, when the latter was the immediate cause of death; rheumatic heart disease plus other conditions contributory to the cause of death; other immediate causes of death plus rheumatic heart disease; rheumatic heart disease plus pregnancy when death was due to the combination of the two; immediate cause of death cerebral embolism plus rheumatic heart disease when the source of the embolus was the heart — no recent myocardial infarction; immediate cause of death pulmonary embolism plus rheumatic heart disease when the source of embolus was the heart — no recent myocardial infarction; and immediate cause of death embolism to other organs or areas plus rheumatic heart disease when the source of the embolus was the heart — no recent myocardial infarction.

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§Deaths attributed to rheumatic fever are generally considered to be due to active rheumatic heart disease.

It has been increasingly difficult to determine from the death certificates prior to 1920 what was and was not rheumatic heart disease. For this reason our study includes only records of the years 1920–1948. The number of deaths and death rates does not necessarily coincide with death rates of the Bureau of Vital Statistics of the Connecticut State Department of Health, since the judgment, interpretation and classification of the actual death certificates in this study may have varied in some cases from the state and city compilation of these statistics.

The change in coding procedures for deaths that was effected in 1938 would not have influenced the data reported here, as causes of death were determined directly from the death certificates and not from coded lists.

TABLE 1. *Population Data for New Haven, Connecticut, 1920, 1930, 1940 and 1950.*

| YEAR | TOTAL POPULATION | 2-24 YEARS* | OVER 24 YEARS |
|---|---|---|---|
| 1950 | 163,344 | 61,053 | 95,968 |
| 1940 | 160,605 | 60,162 | 96,739 |
| 1930 | 162,655 | 69,316 | 88,521 |
| 1920 | 162,537 | 69,176 | 86,327 |

*Estimated by deducting twice the enumerated population under 1 year from the enumerated population under 25 years.
Source: 14th, 15th and 16th Census of the United States and the 1950 census of population — preliminary counts.

Crude annual age-specific mortality rates were calculated in the usual manner. Population figures used in the determination of rates are shown in Table 1. In the absence of exact population figures for each year, a graph was constructed indicating population trends for the two age groups (2 to 24, and 25 or more years of age), and estimates of the age groups for each year were thus used for calculating annual age-specific death rates. It is apparent from Table 1 that although changes in total population for New Haven from 1920 to 1948 were small, changes in the age distribution were appreciable and in keeping with those in the nation at large, with a tendency for the percentage of the population in the first three decades of life to diminish and for the percentage in the older age groups to increase.

A distinction was made between rheumatic heart disease plus heart failure in two age groups, that is, 2 to 25 years and more than 25 years. In patients 25 years of age or less, heart failure is generally considered to be associated with and due to active rheumatic heart disease, whereas in persons over 25 failure is not usually associated with active rheumatic heart disease. Exceptions did occur, particularly in the age range 25 to 35, in which death was due to active rheumatic heart disease in a small number of cases. These were not included among those 25 years or older who died of rheumatic heart disease plus failure. It has been assumed for

TABLE 2. *Number of Deaths Due to Rheumatic Heart Disease and Its Complications.*

| YEAR | R.H.D.* PLUS FAILURE OVER 25 YEARS | R.H.D. 2-24 YEARS | R.H.D. PLUS S.B.E.† S.B.E. CAUSE OF DEATH | R.H.D. PLUS PREGNANCY | R.H.D. PLUS OTHER SECONDARY CAUSES OF DEATH | OTHER PRIMARY CAUSES OF DEATH PLUS R.H.D. | CEREBRAL EMBOLISM PLUS R.H.D. | PULMONARY EMBOLISM PLUS R.H.D. | EMBOLISM TO OTHER ORGANS OR AREAS PLUS R.H.D. |
|---|---|---|---|---|---|---|---|---|---|
| 1948 | 18 | 0 | 1 | | 4 | | 3 | 1 | |
| 1947 | 14 | 2 | 2 | | 4 | | 1 | | |
| 1946 | 17 | 5 | 1 | 1 | 2 | | | 2 | |
| 1945 | 13 | 0 | 3 | | 5 | 3 | 2 | 4 | 1 |
| 1944 | 16 | 1 | 3 | | 4 | 7 | 3 | 2 | |
| 1943 | 21 | 3 | 2 | 1 | 8 | 5 | 2 | | |
| 1942 | 11 | 4 | 5 | 1 | 8 | 5 | 2 | 2 | 4 |
| 1941 | 14 | 3 | 4 | | 4 | 5 | 1 | 1 | 1 |
| 1940 | 15 | 11 | 1 | 1 | | 3 | 3 | | 1 |
| 1939 | 18 | 9 | 4 | | 3 | 2 | 3 | | 1 |
| 1938 | 14 | 11 | 6 | | 5 | 6 | 1 | 2 | |
| 1937 | 12 | 4 | 6 | 1 | 1 | 9 | 4 | 1 | 2 |
| 1936 | 17 | 10 | 3 | 1 | 4 | 6 | 1 | 1 | |
| 1935 | 13 | 6 | 2 | | 4 | 7 | 1 | | 2 |
| 1934 | 19 | 5 | | | 5 | 7 | 2 | 1 | 4 |
| 1933 | 13 | 14 | 1 | | | 5 | | | |
| 1932 | 21 | 7 | 4 | 1 | 5 | 3 | 1 | 1 | 1 |
| 1931 | 20 | 9 | 1 | | 4 | 1 | | | |
| 1930 | 20 | 16 | 2 | 1 | 5 | 5 | 1 | 1 | |
| 1929 | 9 | 9 | 2 | | 5 | 2 | 1 | 1 | 1 |
| 1928 | 16 | 11 | 6 | | 7 | 5 | 2 | | 1 |
| 1927 | 19 | 8 | 5 | | 5 | 7 | 3 | | |
| 1926 | 16 | 17 | 5 | 1 | 3 | 2 | 2 | | |
| 1925 | 18 | 16 | 4 | 1 | 1 | 1 | 2 | 1 | 1 |
| 1924 | 17 | 18 | 3 | 1 | 6 | 2 | 3 | 1 | |
| 1923 | 16 | 14 | 2 | 1 | 5 | 1 | 1 | | |
| 1922 | 13 | 13 | 2 | 2 | 5 | 1 | 1 | | 1 |
| 1921 | 18 | 21 | 4 | 1 | 5 | 1 | | | |
| 1920 | 17 | 16 | 2 | | 5 | 3 | | | |
| Totals | 465 | 263 | 86 | 15 | 118 | 108 | 46 | 22 | 21 |

*R.H.D. — Rheumatic heart disease.
†S.B.E. — Subacute bacterial endocarditis.

purposes of this study that the distinctions made are generally correct, and all deaths recorded as due to rheumatic fever (and all these were between the ages of 2 and 20 years) have been considered deaths due to *active* rheumatic heart disease.

## Results

One thousand one hundred and forty-four deaths due to rheumatic heart disease or its complications occurred in the city of New Haven from 1920 to 1948. Under the 9 categories listed previously, the number of deaths that occurred each year are recorded in Table 2. Age-specific death rates due to rheumatic heart disease plus failure in the age group above 25 years and the group 2 to 24 years of age inclusive are shown in graphic form in Figure 1. Less than 10 per cent of all deaths due to rheumatic heart disease plus failure above the age of 25 were recorded as associated with active rheumatic heart disease.

There were yearly fluctuations in the death rates in the two groups in which death was attributed to rheumatic heart disease plus failure; in the 25-or-older group no significant trend could be seen. In sharp contrast, in the 2-to-25 group (those who in most instances died of active rheumatic heart disease) the trend was consistently downward from a high of 21 deaths in 1921 (0.3 per 1000) to no deaths in 1945 and 1948. This was one of the most significant findings of the study. In the five-year period 1920–1924 there were 81 deaths due to active rheumatic heart disease in persons 25 years of age or less; in the five-year period 1944–1948 there were only 8.

The number of deaths listed under the other classifications did not show any noteworthy change. Deaths due to bacterial endocarditis superimposed on rheumatic heart disease fluctuated from year to year, but showed no consistent trend, either up or down, and the numbers dying of this complication of rheumatic heart disease did not change significantly after the advent of chemotherapy and antibiotics. The number of deaths due to rheumatic heart disease plus other conditions contributing to the cause of death or from other immediate causes plus rheumatic heart disease cannot be said to show any marked change. The majority of deaths due to embolism associated with rheumatic heart disease were cerebral. Pulmonary embolism was less common, as were emboli that lodged in the mesenteric or common iliac arteries at the bifurcation of the aorta or in the coronary arteries. Two in-

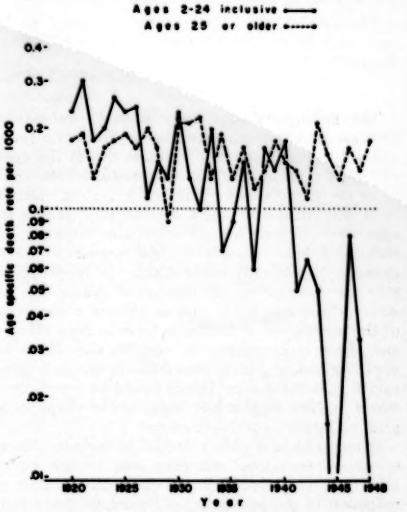


FIGURE 1. *Age-Specific Death Rates (per 1000) for "Rheumatic Heart Disease Plus Failure" in New Haven, Connecticut, for the Years 1920–1948.*
*Rates are calculated for two age groups: 2–24 inclusive, and 25 and older. The downward trend in the former over the past 15 years is apparent.*

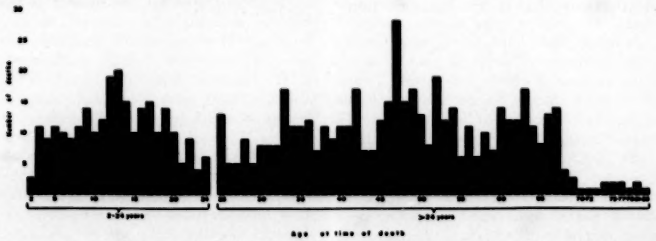

FIGURE 2. *Age at Time of Death for Rheumatic Heart Disease in New Haven, Connecticut, for the Years 1920–1948.*

stances of the latter complication were recorded. There were 15 deaths due to rheumatic heart disease in pregnant women.

### *Age at Time of Death*

The ages at death for the two groups 2 to 24 years and 25 or older are shown in graphic form in Figure 2.

## Discussion

The findings of this study seem to indicate a decrease in the death rate due to rheumatic fever and active rheumatic heart disease within the ages of 2 to 24 years in New Haven from 1920–1948, that is, in the juvenile and young adult groups. Death rates for rheumatic heart disease plus failure in ages over 25 and for all other complications of rheumatic heart disease do not appear to have changed significantly since 1920. It is probable that the decrease in the number of deaths due to active rheumatic heart disease reflects a lowering of the prevalence of rheumatic fever in New Haven and there is no reason to suppose that there is anything unique about New Haven, for it is presumed that these same trends would be found elsewhere in New England at least, and perhaps in a great many places in this country.

It might be said that a decline in the prevalence of upper-respiratory infections due to the beta-hemolytic streptococcus would eventually result in a reduction in the prevalence of rheumatic fever and rheumatic heart disease, since evidence is steadily accumulating to support the hypothesis that rheumatic fever is preceded by such an infection. Certainly it is clear that one form of beta-hemolytic streptococcal disease, scarlet fever, is steadily declining in incidence and severity. Whether other forms of streptococcal infection not accompanied by a rash are decreasing is not known. There seems to be little doubt that one factor that might influence the host's exposure rate to beta-hemolytic streptococcal infections, that is, the home environment, including crowding and housing, has steadily improved in the past fifty years or even in the thirty years covered by this study.

## Summary and Conclusions

A study of mortality due to rheumatic heart disease and its complications in the city of New Haven for the years 1920 to 1948 inclusive is reported.

During this period 1144 deaths due to these causes occurred.

A noteworthy trend occurred in the age group of 2 to 24 years, in which there was a steady decline in the death rate for rheumatic heart disease.

No consistent change could be noted in death rates due to rheumatic heart disease plus heart failure in ages over 25 or in death rates for any of the complications of rheumatic heart disease.

Appreciation is expressed to the Office of Vital Statistics of the City of New Haven for making available the original death certificates. Mr. John T. Coleman, Registrar, and Miss Anna V. Daly, Assistant Registrar, were particularly helpful.

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## MEDICAL PROGRESS

# PLASTIC SURGERY: HARELIP AND CLEFT PALATE (Concluded)*

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**With a Section on Speech Therapy by**

HENRY C. BRITTON, M.A.§

### OPERATIVE PROCEDURES IN CLEFT PALATE

The evidence for and against early closure of the palate has been discussed. The practice among most surgeons in New England is to perform the operation between the ages of eighteen and twenty-four months. Good speech, which is the prime objective in palate closure, depends on a soft palate of sufficient length and mobility that closure of the nasopharynx is possible by virtue of contact between the soft palate and the posterior and lateral pharyngeal walls. Mobility of the soft palate and the nasopharynx is most important in establishing normal speech. Constant motion of these structures in conjunction with the formation of each syllable can be seen by direct observation during speech. The rare adult with an untreated cleft palate and normal speech shows hypertrophy and hypermobility of the nasopharyngeal muscles that compensate for the deficiency of the palate.

For purposes of description, Veau[34] had divided the form of cleft palate into four types (Fig. 6). Type I involves the soft palate only, Type II the soft and hard palate up to the alveolar process, Type III the soft and hard palate and the alveolar process and Type IV soft and hard palate and a double cleft of the alveolar process.

The Dieffenbach–Warren operation for cleft palate is still satisfactory as a routine procedure. In this operation mucoperiosteal flaps are elevated from each palatal process. The palatine arteries are preserved. Through the incisions that extend behind each maxillary tubercle the hamular processes are identified and fractured medially. This releases the tendons of the two tensor palati muscles, which allows greater relaxation of the soft palate and converts the tensor muscle into a levator. Section of the palatal aponeurosis at the posterior border of the palatal process is the final step in mobilizing the palate. The medial surfaces of the soft palate are incised and the palate closed with interrupted stitches. If the cleft extends through the alveolar process mucosal flaps are mobilized from the bone to leave the bony surfaces in apposition, and the flaps are accurately sutured together across the defect and also in the buccal fornix.

Closure of the soft tissue in the upper buccal fornix and at the alveolus is not emphasized by some authors. Small perforations can be very annoying, even though covered by a denture. Mucus and bits of food find their way into them, and some patients are conscious of the escape of air through these fistulas. It is quite possible to close these openings effectively in conjunction with palate repair.

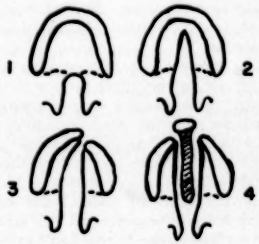


FIGURE 6. *Types of Cleft Palate.*

Speech results have proved least satisfactory in Veau Types I and II. There is often insufficient tissue in each of these to secure proper length by merely closing the cleft. Several valuable improvements on the basic procedure have been made. These have served to reduce the incidence of failures. Wardill,[33] Dorrance[20] and Brown[35] have each described methods of elongation of the palate by shifting tissue from the hard palate posteriorly. In clefts of Type II Wardill elevates triangular mucoperiosteal flaps from either palatal process; these are sutured together in the midline as the palate is displaced backward. In Types I and II both Dorrance and Brown elevate the mucoperiosteum of the hard palate and move it backward with the soft palate. Dorrance cuts the two palatine arteries at the first of two stages and puts a skin graft on the upper surface of the hard palate. When

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the "push-back" is done later no raw surface is left on the floor of the nose. Brown does a one- or two-stage "push-back" operation in Type I clefts but does not cut the arteries. In a Type II cleft a primary closure is done and the anterior palate is later split so that the final procedure is converted into a Type I "push-back." Conway[36] has suggested cutting away the bone about the palatine foramen to increase the mobility of the palate flap. This may answer Dorrance's insistence on cutting the artery as a preliminary to the "push-back" procedure.

Other methods of improving the muscular closure between the posterior wall and the palate include palatoplasty (Wardill[22]) and pharyngeal flap.[37] In the former, Passavant's cushion is further elevated by converting a transverse incision into a vertical closure. In the latter a flap from the pharyngeal wall is sutured to the posterior margin of the soft palate, creating a bridge against which the lateral constrictors of the pharynx can act. A third method is suture of the posterior pillars to reduce the width of the pharyngeal opening. Reduction in the mobility of the palate is the principal disadvantage of this procedure.

Increasing interest has been shown in the raw area usually left on the upper surface of the palate after repair. This concern seems reasonable, as the consequent scarring may interfere with the normal development of the maxillas and expansion of the dental arches and may reduce the mobility of the palate and the effectiveness of a "push-back" procedure. A number of technics for minimizing the scarring have been suggested. A skin graft on the upper surface of the palate flap has been mentioned. Also mentioned has been closure of the anterior cleft with a flap from the vomer at the time of the lip repair or as a preliminary step to final palate repair. The mobilization and suture of flaps of nasal mucosa from the upper surface of each palatal process are also possible at the same time as the palate repair. This technic is commonly practiced in Great Britain. Finally, mobilizing each palatal process and its two mucosal coverings by chiseling them free from the alveoli prior to midline suture of the mucosa will eliminate a raw surface on the floor of the nose. Such gross disturbance of the bony structure hardly seems warranted, but reports of results suggest that it may be a useful method.

### Preoperative and Postoperative Care[38]

Undernutrition and anemia are not uncommon in the patient with a harelip and cleft palate. These evidences of feeding difficulties must be corrected before operation on the lip is contemplated. A better regulated diet, more frequent feedings or transfusions may be necessary. Postoperative bleeding is one of the dangers of the operation; the clotting time must be checked before operation. The usual routine examination must be done, with special search for other anomalies.

After both the lip and the palate operation bleeding must be looked for. The suture line of the lip, which may be covered with a dressing for twenty-four to forty-eight hours, should then be kept clean and dry until the skin sutures are removed on the fourth or fifth day. Likewise, all food particles and crusts of blood should be washed from the palate sutures and the mouth after each feeding. In a child the arms should be held fully extended with splints to avoid his injuring the healing tissues. In no circumstances should anything hard (such as a tongue depressor or toast) be allowed in the mouth.

Otherwise routine supportive measures and care should be given these patients just as to any postoperative patient.

### Feeding

The infant with a cleft lip and palate cannot take its feedings normally. He is unable either to squeeze a nipple or to maintain suction when swallowing. Feeding in patients with more severe clefts is a tedious process. Before the lip is closed the baby usually can best be fed with a large dropper to which a short rubber tube is attached. Establishing this method of feeding as a habit is desirable preoperatively, as it is routinely used postoperatively for two or three weeks until the lip is healed.

Abnormally large rubber nipples, nipples with large holes and nipples with a flat pad on one side to cover the cleft palate have all been found useful after the lip is repaired or in babies with a cleft palate only. The problem is one of getting the feeding down without choking the baby and with as little swallowing of air as possible. Much patience is required to persuade the infant to take the full amount. At times more frequent feedings may be desirable, but tube feedings should be avoided.

Breast milk or a formula may be used. The quantity should be the normal amount or possibly slightly more than normal for the child's age and weight. The infant with a cleft palate may eat better if weaned early and fed strained foods. This is by no means essential but may simplify the infant's care. At the time of the palate repair the child who is accustomed to drink from a cup is easier to feed than one who has not been weaned.

After the operation for cleft palate a liquid diet and later a soft diet are essential for several weeks until the palate is fully healed. Normal feeding can then be resumed.

### Dental Problems

Clefts of the alveolus and palate present special problems to the dentist, orthodontist and prosthodontist. The teeth in many of these children are "soft" and subject to severe caries. Those that lie adjacent to the cleft or in the premaxilla may

have insufficient bone about them to be moved safely. In each of these circumstances extraction may be necessary. It is desirable to preserve the teeth if at all possible, to stimulate development of the jaws and to provide anchorage for orthodontic appliances. Early orthodontic attention to these teeth can maintain proper orientation of their inclined planes and reduce the narrowing of the maxillary arches. The work of the orthodontist must start early and may continue into puberty.

Dentures or more elaborate appliances are frequently needed in patients with an alveolar cleft. Missing or extracted teeth must be replaced and functional occlusion re-established. Retraction of the upper lip in the absence of teeth or of the premaxilla requires the construction of a denture that will press the lip forward. The formation of an upper buccal fornix, with or without a skin graft, and other soft-tissue restoration may have to be a preliminary step. If insufficient palatal tissue is available or if extensive scarring from unsuccessful procedures makes further surgery impossible, an appliance is necessary to cover the roof of the mouth. This appliance may have attached to it a hollow cast bulb that partially occludes the nasopharynx and aids normal closure. The technic of closing the palate with a prosthetic appliance antedates the surgical closure. In fact, its development was stimulated by the consistent failure of operative procedures before the present-day methods were devised. V. H. Kazanjian[39] of Boston and the late John J. Fitz-Gibbon[12] of Holyoke have made important contributions to the more effective use of prosthetic appliances for the patient with a cleft palate. The former has combined a broad understanding of the possibilities of soft-tissue manipulation with an expert knowledge of the possibilities of prosthetic appliances. The latter had an extensive experience in the construction of these appliances for the restoration of facial contours and for improving speech.

## Speech Therapy

HENRY C. BRITTON, M. A.

The functions of the speech therapist may be defined in terms of three concepts — the objectives of the treatment program as a whole, the nature of the speech problems and the administrative organization of the total program. The objectives of treatment as a whole seem to be in transition from an emphasis on optimal lip and palate functioning to a concern for the rehabilitation of the child as a whole, as a member of society. With this change — from rehabilitation of the cleft palate to rehabilitation of the child with cleft palate — there has been a corresponding change in speech therapy.

The typically defective speech of children with cleft palate is generally considered so serious a handicap psychologically, socially and vocationally that its elimination is an essential goal of the treatment program. However, there is not an equally general agreement concerning the causes of this defective speech.

Superficially, the relation between cleft and speech defect seems uncomplicated. The speech is nasal because there is a cleft in the palate, with more or less of an opening between the oral and nasal cavities at all times. The speech will no longer be nasal when adequate palatal functioning or its prosthetic equivalent is obtained. If after treatment speech is still nasal, further surgery, prosthodontia or speech therapy, or a combination of these, is indicated. Refinements of technic in the three specialties should result in a closer approximation of normal velopharyngeal function and therefore should result in better speech.

There are two reasons for questioning the validity of the assumptions that nasality is a direct result of the palatal cleft and that normal speech should come with adequate velopharyngeal functioning. First results of treatment based on these assumptions have not been satisfactory.[40] Secondly, the two assumptions are difficult to reconcile with clinical observation and with published experimental data.

If these assumptions were valid, it would follow that the degree of nasality should vary directly with the size of the cleft. This hypothesis is not verified by observation. Occasionally a child is seen with a large palatal cleft but without nasal voice quality; more often a child may have apparently adequate palatal functioning but conspicuously nasal speech.* Furthermore, most children with a palatal cleft can make at least a few non-nasal speech sounds. In the course of studying normal palatal functioning it has been found that the average speaker may make some of the non-nasal speech sounds with slightly lowered velum and that the velopharyngeal opening can be approximately 20 sq. mm. in cross-section before nasality is detected in the voice.[41, 42] Similar observations have been made of the voice in singing.[43] The velopharyngeal opening may be of any size without producing nasality, provided that the minimal tubular area of the anterior nasal passages is greater than that of the posterior opening. One physiologic basis for nasality or non-nasality, therefore, is the relative size of the anterior and posterior nasal openings.[44] Nasality may also result if the labial opening is smaller than either the anterior or posterior narial one.[44]

A satisfactory physiologic concept of nasality is not at present available, partly because of the complexity of the speech mechanism and partly

*In some instances there may be a normal-appearing palate with the mucous membrane intact but a congenital absence or failure of some of the underlying palatal muscles to unite. This submucosal type of cleft is not often referred to the plastic clinics because the cleft is not recognized as such. Although a split uvula of itself should not cause nasality, it may accompany a submucosal cleft, which does cause it.[11, 40]

because of a complexity of "nasalities."* To date, the types of nasality are best defined as subjective auditory concepts and best measured as acoustic phenomena.[46, 47]

Other speech defects, such as delayed speech or mispronunciations, are often superimposed on the nasality. These may be fostered by the unfortunate, though understandable, tendency of some parents to fail to adjust to the child's cleft. They may consciously or otherwise reject him, or they may "make it up to him" by doing things for him that he should be doing for himself. In brief, they do not treat him as a normal child and therefore fail to provide the stimulation he needs for emotional, social and linguistic growth. One of the essential bases of speech is being talked to and with, frequently and as a part of everyday living. Failure to provide this stimulation can result in delayed or defective speech, just as it could with a normal child.

As implied in the preceding paragraphs, the speech therapist working with children with congenital palatal cleft is beginning to extend his interest to the speech mechanism as a whole, rather than limiting it primarily to palatal function.

As a participant in what is frequently called, in spite of rather incongruous connotations, the "cleft-palate team," the speech therapist shares in the extending of treatment from the palate to the child as a whole. Successful rehabilitation is then seen as the product of the joint efforts of all concerned, — in particular those of the child and his parents. The same may be said of the speech aspects of the rehabilitative process. Speech is the concern of each member of the team.

The place of the surgeon in influencing the speech pattern has been discussed in the first part of this paper. The speech therapist supplements the operation by working toward the related goals of improved speech and improved appearance. The speech aspects have been briefly considered in the preceding paragraphs. In improving appearance, the therapist tries to encourage more effective use of the facial muscles (particularly those of the upper lip) in speech. Some of these muscles tend to contract minimally or even not at all. In the case of a scarred lip, the relative immobility tends to draw attention to that part of the face. Normal speech also postulates normal appearance while speaking.

Whatever the orthodontist may do to improve masticatory function and facial appearance will aid in the attaining of normal speech. Much concern has recently been evinced with retardation of the growth of the middle third of the face as a result of early or inappropriate surgical closure of the palatal cleft.[24] One solution proposed is the postponement of closure until the child is four years old or more. Although they recognize that the problem of disproportionate rates in facial growth must be solved if the child is to be rehabilitated, speech therapists cannot be expected to be enthusiastic about late closure, as such. After the age of three, secondary speech and personality defects compound the rehabilitative effort.[40] If delayed surgery is advocated, it would seem that increased consideration should be given to temporary prosthesis or to speech therapy before the operation.

The speech therapist is interested in the otologic aspects of rehabilitation. Surprisingly little is published concerning middle-ear involvement as related to cleft palate. On the basis of unpublished data from several sources, including a series of 75 cases attended by the writer during the last year, it can be stated conservatively that at least 25 per cent of the children with cleft palate may be expected to have ear involvement severe enough to require special consideration in therapy. This is mostly of the nature of a middle-ear infection and hearing loss or both.[48-50] Children with a congenital palatal cleft seem more susceptible to upper-respiratory infections in general. Fluctuations in hearing acuity are sometimes observed, the hearing being poorer when an upper-respiratory infection is present and when the humidity is high. Hearing acuity has been improved in some cases by removal of tonsils and adenoids.

The accepted view concerning the removal of tonsils and adenoids in patients with an operatively closed palatal cleft is that removal should be avoided if possible. Excluding other reasons for not removing them, it is thought that the gap between palate and pharyngeal wall might in their absence be too wide for closure in speech. In the face of what seems to be unanimous opinion, I can only state that I have not as yet seen an instance of ordinarily careful excision of the tonsils or adenoids that affected the speaking voice adversely. In most cases there was improved pharyngeal motility and a resulting decrease in nasality.

A poorly nourished or sickly child with cleft palate has a poor prognosis for obtaining normal speech unless a change for the better occurs in his physical status. It has been observed in the clinics that of children seen prior to operation the more active ones tend eventually to have better speech results.

*An attractive hypothesis, with some experimental support, can be developed by considering the two types of open nasality — the active and the passive — as acoustic by-products of two ways of adjusting to the disturbance of nasal functioning associated with cleft palate or cleft lip or both. Normally, the nose has an important function in the regulation of the amount of air taken into the lungs. Rohrer[45] has stated that the nasal canal offers 50 per cent of the total tubular resistance to the inspired air. If the nasal canal is too narrow (causing mouth breathing) or too open, compensatory constrictions will be made by some of the parts of the speech mechanism—lips, back of the tongue, muscles of the oropharynx, epiglottis, false vocal bands and sometimes the nasal constrictors or even the repaired velum. These constrictions have been identified as factors in nasality.[46] This may be described as the active type of compensation. In the passive compensatory type, the demands of the organism for increased respiratory rate and volume in activity are minimized by reducing the need for activity. In these children there is a palatal hypotonicity as part of a general pattern of muscular hypotonicity. There are sometimes in the active, and usually in the passive, disturbed coordinations in breathing during speech. Attention to these, combined with procedures designed to decrease the previously mentioned constrictions, has been very effective in some cases in decreasing open nasality.

Consideration of the child's personal and social adjustments may be critical in speech therapy. If there is a poor adjustment in either area, the efforts of all concerned may not result in improved speech until the maladjustment is eliminated. The social worker and the psychiatrist can have an essential part in preparing the child and the family for understanding and acceptance of the need for speech therapy. The fact is sometimes overlooked that a child cannot be "taught" until he is able and willing to learn. Every clinician is probably familiar with the child whose speech is conspicuously more nasal when he doesn't get what he wants when he wants it. Nasality has been known to decrease rapidly when the patient reaches adolescence and his voice is pitched lower, but it has been known to decrease still more rapidly with increased interest in a vocation or in the opposite sex.

It has seemed to the writer that one of the most important functions that those on the team can fulfill is to give the child encouragement toward greater self-respect as a member of society, simply by considering him in fact and by implication as essentially normal. Without this attitude speech achievement may be poor in spite of otherwise excellent therapy; with it even ordinary or inadequate therapy may be successful.

## Summary

It is hoped that this paper will furnish a greater interest and understanding of the many and varied problems involved in the care of infants and children with harelip and cleft palate.

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# CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

## Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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## CASE 37321

### PRESENTATION OF CASE

A fifty-two-year-old woman entered the hospital complaining of severe headache.

The patient had had high blood pressure and headaches — mainly left occipital — for thirteen years. Seventeen days before admission she began to have dizzy spells accompanied by a buzzing and ringing in the ears. She also staggered slightly to the left. A week after the onset the headaches increased in severity. The patient became nauseated and repeatedly vomited ingested food, without blood. Five days before admission she entered another hospital, where a lumbar puncture showed high pressure; a Papanicolaou smear on the spinal fluid was reported as "positive." During the five-day period that she stayed in that hospital she was nearly always in bed and drowsy. There was no history suggestive of a pulmonary, breast, rectal or gastric malignant process. There were no changes in speech or articulation and no convulsions.

Physical examination revealed an obese, flushed, drowsy woman who responded well when addressed. Examination of the eyes showed ++ papilledema bilaterally and hemorrhages that were limited to the optic disks. Extraocular movements showed weakness of the left lateral rectus and there was a questionable left-lower-facial weakness. The tongue protruded in the midline. Sensation was normal. The tendon reflexes were hypotonic. The heart rate was 58, with a Grade I systolic murmur. The lungs were clear and the abdomen was negative.

The blood pressure varied between 215 systolic, 90 diastolic and 230 systolic, 120 diastolic.

Examination of the blood revealed a hemoglobin of 15 gm. and a white-cell count ranging from 11,700 to 17,000. The urine was normal. Lumbar puncture showed a clear, colorless fluid under a pressure equivalent to 600 mm. of water. The protein was 83 mg. and the sugar 82 mg. per 100 cc. There were 150 cells per cu. mm., 50 per cent of which were red cells, 35 per cent small mononuclear cells and 15 per cent round cells ranging from 30 to 80 microns in diameter. Smears and culture were negative for bacteria and yeast organisms. An electroencephalogram showed a diffusely abnormal record with no evidence of focal disorder. Her mental status at times was adequate for cortical-function tests, which were normal. X-ray films of the skull showed thinning of both posterior clinoid processes. The pineal body lay in the midline. A film of the chest was negative.

On the fifth hospital day she became comatose and completely unresponsive to stimuli. A questionable weakness of the right-facial muscles developed and the right lateral rectus was thought to be weak. Burr holes were made. The ventricular fluid contained 100 cells per cu. mm., with 80 per cent red cells, 12 per cent mononuclear cells and 8 per cent of the large round cells noted previously. The patient went progressively downhill, and the temperature rose to 102°F. and the white-cell count to 22,300. A subtemporal decompression on the left side was carried out in an attempt to relieve the high intracranial pressure, which was equivalent to 600 mm. of water on both spinal and ventricular taps. A ventriculogram showed that the posterior margin of the fourth ventricle appeared to be displaced anteriorly and upward. A small portion of the aqueduct that was visualized appeared to be angulated as if displaced upward.

On the eighth hospital day an operation was performed.

### DIFFERENTIAL DIAGNOSIS

DR. WALLACE ZELLER*: It is unlikely that this illness could be explained solely on the basis of hypertensive disease. As a matter of fact, I am not sure that the patient had much in the way of serious organic disease associated with the hypertension that she had for thirteen years. There was no evidence of any retinal complication that might have been directly laid to that. The urine was normal, so that it is unlikely that there were any renal complications. There was no evidence of the anemia that may go with chronic renal disease. The spinal-fluid findings are more consistent with other diagnoses and leave me in considerable doubt.

The history is that of a rather rapidly progressing illness in which attention is directed to the central nervous system by the patient's complaints and by the rather sudden onset of rapidly increasing intracranial pressure manifested predominantly by headache, vomiting and papilledema. What type of intracranial disease was this? Certainly it is definite, judging from the history, that brain tumor was considered. As in all such cases, a metastatic tumor was thought of, and a chest film was taken in order to be sure that there was no evidence of

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primary carcinoma or a metastatic nodule in the lung field.

Dr. Wyman, would you show the x-ray films?

Dr. Stanley M. Wyman: I am sorry that the films are not available.

There was an anteroposterior film taken with the patient lying down and a lateral film that was taken at the same time. These were of indifferent quality because of the patient's size and condition, but they seemed to be normal.

Dr. Zeller: A small tumor of the lung could be missed in an examination of that type.

Dr. Wyman: Yes; it could.

Dr. Zeller: I should like to ask: What is the significance of the thinning of the posterior clinoid processes noted in the skull films?

Dr. Wyman: Thinning of the posterior clinoids is one of the earliest and commonest signs of increased intracranial pressure, and the lesion may be near to or far from the sella turcica. It has no localizing value. There was a suggestion that the left posterior clinoid was thinner than the right, but that may have been a developmental anomaly.

Dr. Zeller: It is interesting that the patient's symptoms were mainly left-sided at first. There seems to be little doubt that the ventriculogram showed real abnormalities.

Dr. Wyman: The description in the record makes one worry about a posterior-fossa lesion.

Dr. Zeller: Such a lesion could involve the brain stem and cause some difficulty in drainage through the aqueduct because of upward dislocation. The other thing that is extremely intriguing about this case is the description of round cells that ranged in size from 30 to 80 microns in diameter. I wonder if it is possible at this time to learn any more about these cells?

Dr. Tracy B. Mallory: Eighty microns would be ten times the diameter of a red cell. I am not sure I know of any mononuclear cells of that size.

Dr. Zeller: Would you be inclined to consider the extreme size an artifact, Dr. Mallory?

Dr. Charles S. Kubik: These cells were actually measured; they were larger than any cells commonly seen in a histologic preparation.

Dr. Zeller: Were any mitotic figures or multiple nuclei seen?

Dr. Kubik: I do not think so.

Dr. William H. Sweet: We asked the opinion of numerous cytologists and cellular pathologists in the greater Boston area and received quite a variety of opinions concerning what these structures may have been. They had a homogeneous, darkly staining sphere in their centers surrounded by a large area of structureless transparent material with a clear-cut limiting membrane at the periphery. They looked something like a gargantuan torula organism. There was no unanimity about whether they were actually cells or whether they represented some form of yeast or fungus. I think the minority impression was that special stains for a capsule showed one to be present and suggested that the structure was a cryptococcus of some kind; the majority thought these large objects were cells.

Dr. Mallory: Yeast of this size would be even more extraordinary than a cell of this size.

Dr. Zeller: Did the Cytology Laboratory look at these?

Dr. Sweet: They were reported negative for carcinoma.

Dr. Zeller: I understand that it is extremely difficult to use the Papanicolaou technic with spinal fluid. Usually there are so few cells that it is likely to be unsatisfactory.

Dr. Sweet: Enormous numbers of these cells were seen in the routine spinal-fluid sample.

Dr. Zeller: It is interesting that the question of yeast infection did come up. I believe it has to be included among the first group of diseases that I should consider. This group, of course, includes other infections. I wonder if this picture could be produced by some type of tuberculosis, syphilis or yeast infection. I think it is quite unlikely, in view of the unusual type of cells described in the spinal fluid. There was a decrease of the spinal-fluid glucose, and this fact would not be consistent with tuberculous or yeast infection. I believe the cryptococcus neoformans itself is 6 or 7 microns in diameter, but it has a tremendously big capsule, which may be 5 times the diameter of the organism itself. If the capsule were mistaken for cytoplasm, the organism might then be in the lower range of size of these questionable cells.

I have compared two cases of torula meningitis reported from the Mount Sinai Hospital[1] with the case under discussion. It is amazing how similar they are. Duration of the illness in the two reported cases was brief. At autopsy chronic inflammatory tissue was found about the medulla, producing internal hydrocephalus. In one instance it was extremely difficult to demonstrate the organism, and it was only done at autopsy. The patient in today's case was operated on on the eighth day. It may be that the cultures, which were negative preoperatively, did become positive after that time. If they had, I believe I should have been given the information. Certainly it takes up to two weeks to obtain a positive culture in some instances. I do not believe the data are adequate to support a diagnosis of torula meningitis.

I have thought of other types of parasitic disease, such as cysticercosis, echinococcic cyst and schistosomiasis, only to throw them out. There are no good data pointing to the presence of a single inflammatory tumor mass. I believe brain abscess too, may be discarded. I do not know of any primary tumor of the brain stem that has ever given rise to spinal-fluid findings of this sort. The most

common primary brain tumors in this site are glioma of the pons and acoustic neurinoma. The progress of the disease seems to be much too rapid for the latter, in which the history usually goes back for some years. In this case it was only a matter of days.

I have tried to get more information about the occurrence of tumor cells in the spinal fluid. Willis's *Pathology of Tumors*[2] has interesting data on this subject. The author states that glioma cells have never been found in the spinal fluid during life. He believes that cells of medulloblastoma would be most likely to appear in this fashion. Such cells would be very difficult to distinguish from lymphocytes. Cells from many cases of carcinoma, however, have been identified in the spinal fluid. In addition, cells from a thymic tumor in a child, a round-cell ocular sarcoma and probably a pituitary adenoma have been similarly demonstrated. Willis states that the site of invasion of the cerebrospinal-fluid spaces by carcinoma is often evident. Not infrequently, however, disseminated carcinomatosis of the meninges has been present without any demonstrable metastatic growth in the brain at all. The fluid-borne metastases tend to be more abundant about the base of the brain and in the spinal canal, particularly on the dorsal aspect. They are also found in the caudal region.

If I am going to accept the diagnosis of carcinomatosis on this somewhat tenuous evidence, where could it possibly have come from? The chest was not too adequately examined. It is possible that it was the source of a primary growth, with metastatic involvement of the cerebrospinal-fluid space. The gastrointestinal tract is a good possibility. We have nothing contributory in the history or the examination, but because of the circumstances it was not possible to examine the patient very thoroughly. I would certainly think strongly of the pancreas as the site of a tumor that would be unsuspected in the primary location and appear first as a metastatic lesion in the brain or cerebrospinal-fluid space. Also to be mentioned though not discussed, because of the paucity of data, are primary tumors of the stomach, breast, nasopharynx and adrenal glands. There is not much point in discussing each one individually. It is possible in this particular case that at the present time the primary site is still not known.

I believe it is most likely that the diagnosis in this patient was that of metastatic carcinomatosis involving the meninges. It would be interesting and important to collect the data on the spinal-fluid findings in other cases of this type.

DR. TRACY B. MALLORY: I think it is very difficult, from reading the record, to determine much about these cells in the spinal fluid.

DR. JAMES B. AYER: I should like to inject here a possibility that these large cells may have been cells from the arachnoid. Such cells occur in febrile states and in other conditions causing irritation of the meninges. When allowed to remain in the fluid, arachnoid cells may become considerably expanded, but they are likely to contain inclusion bodies. I throw out this suggestion as an alternative possibility to their being cancer cells.

DR. ZELLER: Were they ever as numerous as in this case?

DR. AYER: Often quite numerous.

DR. KUBIK: In what conditions were they observed?

DR. AYER: I have seen them in meningitis, both in man and experimental in animals, and in small numbers in febrile states without too many lymphocytes.

DR. KUBIK: These cells were quite different from anything that I had ever seen in the spinal fluid. Cells of somewhat similar appearance are sometimes found in the fluid of a gliomatous cyst.

DR. MALLORY: As far as these measurements are concerned, they are only accurate in relation to the conditions under which they were made. Cells as seen in smeared preparations always appear much larger than the same cells would in tissue sections. This is due particularly to the fact that in making a smear cells invariably are squashed to some extent. Therefore a spherical cell is flattened out, making it seem much bigger than it would appear in a hanging-drop preparation or, especially, in histologic sections, in which it would be affected by fixation, dehydrating agents and heat.

### CLINICAL DIAGNOSIS

Neoplastic disease, posterior fossa.

### DR. ZELLER'S DIAGNOSIS

Carcinomatosis of meninges.

### ANATOMICAL DIAGNOSIS

*Colloid carcinoma of stomach with metastases to regional lymph nodes, meninges and cerebellum.*

### PATHOLOGICAL DISCUSSION

DR. SWEET: The finding at operation was a little surprising to us. In conformity with the way the x-ray films were interpreted, our preoperative diagnosis was tumor in the posterior cranial fossa. At operation the right-cerebellar hemisphere bulged markedly out of the wound and had on its surface mottled purplish to grayish streaks along the blood vessels that we described as an exudate. That left us wondering what to do. We finally elected, because the cerebellar hemisphere bulged, to remove the posterior protruding portion of it. As we examined the gross specimen, the only abnormal tissue was found along the blood vessels and was seen to be less than a millimeter in thickness; it was

strongly suggestive of an exudate in the cerebrospinal fluid. The wound was closed without further removal of tissue.

DR. MALLORY: The patient died four days after operation. I did not see the brain at the time of post-mortem examination, but as it was described there it was a rather diffuse thickening — clouding of the arachnoid — most marked at the base of the brain but to some extent extending over the cerebellar hemispheres, and the written description sounds not very different from that of a tuberculous

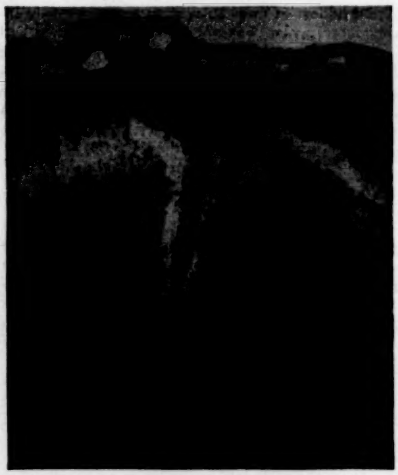

FIGURE 1.

meningitis. However, microscopical sections showed that both the lesion of the cerebellum and the thickening of the arachnoid were due to tumor infiltration that did not look like a primary brain tumor. As the autopsy continued, a peculiar area was eventually found in the stomach — a cleft-like thickening of the gastric wall, including both mucosa and muscularis. Sections of that showed a very typical colloid carcinoma of the stomach with many signet-ring cells showing characteristic nuclear vacuolization. Strangely enough, for a carcinoma of the stomach, no metastases could be found in the liver, and there were no obvious metastases in the lung, either. Many lymph nodes were positive along the lesser curvature of the stomach, but the central nervous system appeared to be the only distant site of metastasis.

DR. KUBIK: We have a lantern slide of the cerebellum (Fig. 1). Even in that the cells were large, though probably not as large as those seen in the cerebrospinal fluid. The subarachnoid space contains tumor cells that extend through the pia into the substance of the cerebellum and also along the perivascular spaces.

I also have a slide from another case of carcinomatosis of the meninges.[3] In this case the primary tumor was not found. There was an invasion of the subarachnoid space with extension through the pia into the spinal and cranial nerves and into the substance of the cord and brain stem. That patient had multiple spinal-nerve and cranial-nerve palsies.

DR. MADELAINE R. BROWN: Was the arachnoid affected only over the cerebellum?

DR. E. PEIRSON RICHARDSON: There was a small amount of thickening of the cerebral arachnoid but nothing as severe as was found over the cerebellum.

DR. THOMAS BALLANTINE: Was there a tumor nodule as well?

DR. RICHARDSON: No tumor nodule.

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## CASE 37322

### PRESENTATION OF CASE

A fifty-nine-year-old house painter entered the hospital complaining of gas pains.

Until approximately one year before entry the patient was said to have consumed from 15 to 20 glasses of beer alternating with one quart of wine per day, probably associated with poor caloric intake. One year prior to admission, however, because of anorexia, he reduced his intake to 1 to 4 glasses of beer a day. Four months prior to entry he noted "gas pains" after eating and at about this same time began to develop weight loss totaling 20 pounds up to the time of admission. One month later he was seen in the Out Patient Department because of two weeks of substernal aching that occasionally spread to the left arm. He seemed to have more cough than usual with the "bronchial asthma" that he had had for years. On physical examination at that time he was slightly cyanotic. The chest was hyperresonant; the heart was not enlarged, and the sounds were very distant. The liver could be percussed 5 cm. below the costal margin but could not be felt. An electrocardiogram was interpreted as probably within normal limits. X-ray films of the chest showed "a small heart and pulmonary emphysema." About two months prior to admission he became constipated, being able to

pass only a small amount of hard brown feces and considerable gas. There was no hematemesis or melena. Three weeks before entry a gastrointestinal series and a barium-enema examination made at another hospital were reportedly negative, but the liver was enlarged. A week later the abdomen became slightly distended, and on the day prior to admission he noted the onset of dependent ankle edema and one-pillow orthopnea.

For thirty-seven years the patient had been a painter, using lead paint. Prior to this he had worked in a mill, exposed to an unknown type of solvent.

Physical examination revealed a cachectic, dehydrated man who showed evidence of some wasting of the upper extremities. The scleras were questionably icteric. The nail beds were cyanotic. The fingernails suggested early clubbing. The chest was hyperresonant to percussion, with poor respiratory exchange to auscultation. The heart was normal. The abdomen was tympanitic and the liver was felt at least 2 or 3 fingers below the costal margin but was difficult to palpate because of ascites. There was slight pitting edema of the lower legs. Rectal examination was negative.

The temperature was 98.6°F., the pulse 110 and the respirations normal. The blood pressure was 140 systolic, 90 diastolic.

The urine had a concentrated specific gravity of 1.016 and contained some bile, as indicated by a Harrison spot test. The urine urobilinogen was 1.1 Ehrlich units in a two-hour specimen. Examination of the blood disclosed a hemoglobin of 15 gm., normal red cells on smear and white-cell counts ranging from 14,800 to 20,250, with a normal differential. Three stools were light tan to brown and ++ guaiac. The total protein was 6.42 gm. with a 1.1 albumin-globulin ratio; the nonprotein nitrogen was 42 mg., the alkaline phosphatase 6.7 units and the serum bilirubin 1.4 mg. indirect and 3 mg. total per 100 cc. The prothrombin time on admission was 62 per cent of normal, rising to 75 per cent twelve hours after 10 mg. of hykinone had been given. Later, however, the prothrombin time fell to 57 per cent. The bromsulfalein retention was 42 per cent in forty-five minutes.

On the second hospital day a paracentesis yielded 3845 cc. of straw-colored fluid with a specific gravity of 1.006 and 350 white cells and 560 red cells per cu. mm. A barium-swallow examination showed a somewhat widened lower esophagus with some curling. The folds in the lower few centimeters of the esophagus were tortuous. A small hiatus hernia was also demonstrated. On the sixth hospital day 3060 cc. of yellow fluid with a specific gravity of 1.004 was removed. A few days later the patient began to develop edema of the genitalia, with difficulty in voiding. On the ninth hospital day a peritoneoscopy was performed. A few hours after this procedure the patient's blood pressure fell to 60 to 80 systolic and the pulse was weak at 100 beats per minute. Twelve hours later the blood pressure became unobtainable. There was no clinical evidence of bleeding esophageal varices. The patient became more icteric and dry and began to breathe deeply, with long expirations. After this he became restless and confused, and died quietly on the thirteenth hospital day.

## Differential Diagnosis

Dr. W. Warren Point*: In short, this was an elderly man with a story of chronic alcoholism for many years. About four months before admission he began to have digestive symptoms, and over that period he lost at least 20 pounds in weight — probably more, which was not counted because he had ascites. He developed signs of rapidly progressive disease, which I believe was liver disease, and entered the hospital with ascites. There was some question about the diagnosis, so peritoneoscopy was performed. I presume the patient went into shock and then into cholemia and died.

May we see the x-ray films?

Dr. Joseph Hanelin: The films of the chest, taken about two and a half months apart, show evidence of the development of ascites, as indicated by the higher position of the diaphragm in the second examination. The lungs are essentially as has been described; there are a few areas of atelectasis in the right lower lung. The heart is relatively small in size. The films of the abdomen confirm the clinical finding of ascites. The precise sizes of the liver and spleen are difficult to determine. However, the liver does appear to be somewhat enlarged, and the spleen may be slightly increased in size, although I cannot define its margins with accuracy. A barium-enema examination is not remarkable. The film made after the barium swallow does show the wide fold in the lower esophagus that was mentioned, and I think the appearance is consistent with small esophageal varices. No statement is made about the stomach, and I have no films of that portion of the examination.

Dr. Point: Do you think the appearance of the varices would suggest that they are perhaps early?

Dr. Hanelin: I think that would be consistent with the relatively small size.

Dr. Point: There is no evidence of any change within the parenchyma of the lung — nothing to suggest lymphatic spread of tumor or granulomatous lesions?

Dr. Hanelin: No; I do not see anything that would indicate malignant disease. Any change in the lung could be attributed to the elevated position of the diaphragm.

Dr. Point: This is a perfectly normal smooth diaphragm, though elevated?

Dr. Hanelin: Yes.

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DR. POINT: A consideration of the causes of liver disease seems to be a good point of departure in this case. I think it best to go over the history to clarify this somewhat confusing picture. The intake of alcohol and the poor caloric intake are certainly consistent with a diagnosis of alcoholic cirrhosis of the liver. However, when he developed anorexia a year before admission he reduced his alcoholic intake considerably. Because he did not lose weight during the next eight months I am forced to believe he probably did eat more food. The anorexia could have been due to alcoholic gastritis, to hiatus hernia, possibly to an early carcinoma of the stomach or even to early manifestations of some intra-abdominal carcinoma from an unknown site.

Four months prior to entry, however, a definite change took place in his condition. The patient began to have gas pains after eating, which does not indicate any specific disease but may have been due to cholecystitis or, if the pains were crampy, possibly to partial small-bowel obstruction, although the picture is not specific for that. A month later he was seen in the Out Patient Department because of substernal aching. I do not know whether it occurred on exertion or after eating. Without anything else to go on, I must say it was probably due to the hiatus hernia. Hiatus hernia may cause substernal discomfort, and it may also cause pain down either arm or anywhere in the chest. Of course, the possibility of angina should be raised, but further examination and the history did not disclose any supportive evidence of this.

The patient also developed more cough. He had "bronchial asthma," which may have been bronchitis or may have been bronchiectasis and emphysema. Patients who become debilitated from any cause and who already have bronchial disease will tend to cough more. I do not think that is of much help to me.

The examination in the Out Patient Department showed cyanosis, which is perfectly consistent with emphysema of a severe degree and is seen in about 5 per cent of cases of Laennec's cirrhosis. The chest was hyperresonant, and the physical signs were consistent with emphysema. This is an important point; although the liver could be percussed 5 cm. below the costal margin it could not be felt. Apparently there was no ascites and the abdomen was not distended; if the liver was enlarged it should have been easily palpable at the time. The constipation of which the patient complained two months before admission was a nonspecific gastrointestinal complaint consistent with many forms of intra-abdominal disease — cirrhosis, carcinoma of the pancreas, carcinoma of the stomach, gallbladder disease and so on. However, it did indicate the progressive nature of the disease. The gas was part of the constipation and the generalized progressive gastrointestinal symptoms. There was no bleeding. The liver was enlarged in the x-ray films made three weeks before admission. A week later the patient began to have abdominal distention, which may have been due to gas and to fluid. This progressed, and edema of the ankles and some orthopnea developed. This is perfectly typical, along with hypoalbuminemia, of decompensation of a cirrhotic liver.

The occupational history is interesting. We know that there are references in the literature to lead poisoning's causing cirrhosis of the liver, but they are not substantiated so far as disease in man is concerned. The unknown solvent to which he was exposed thirty years before admission could have been carbon tetrachloride. However, the past history was nebulous, and exposure occurred thirty-seven years before the final illness. I do not think I can pin anything on that.

On arrival in the hospital the patient obviously had a "mortal" disease, with cachexia and ascites. He did show icterus at that time, which was later confirmed by the van den Bergh reaction and by the Harrison spot test on the urine. A new sign—clubbing — was noted. Clubbing is rare but possible with cirrhosis of the liver or with liver disease of any cause. It is also possible but unusual in pulmonary emphysema in the absence of bronchiectasis, bronchitis or other pulmonary disease. I do not know how significant that was. He had had evidence of bronchiectasis and emphysema for a long time previously. The abdominal examination revealed tympanites and ascites to such an extent that it became difficult to palpate the abdomen. Somehow the examining physician was able to feel the liver at least three fingerbreadths below the costal margin. I assume that the liver had enlarged, and that is borne out by the x-ray films taken at that time. The rectal examination was normal, which along with the barium-enema examination would eliminate lower-bowel carcinoma. He showed no signs of any renal lesion, such as hypernephroma, which may metastasize to the liver.

Urinary bile was present, confirming the jaundice. The urine urobilinogen was 1.1 Ehrlich units in two hours, which is a little above the accepted normal in this hospital; however, it is not definitely abnormal. This is neither for nor against cirrhosis of the liver, metastatic liver disease, infectious hepatitis and so forth, because there are exceptions to all the liver-function tests.

The white-cell count was much elevated, which is extremely unusual in uncomplicated Laennec's cirrhosis of the liver. Apparently no complications had been noted. I do not understand the high white-cell count with a normal differential. The hemoglobin of 15 gm. was probably falsely high because the patient was dehydrated, but at least he was not in the anemic range. It would be a little unusual for far-advanced cirrhosis of the liver to have a hemoglobin as close to normal as this one was. This is one point against cirrhosis.

The light-tan to brown stools indicate that there was at least no complete biliary obstruction. That would be borne out by the low level of the van den Bergh test. The ++ guaiac reactions of the stools on all three occasions make one think there was a bleeding lesion in the gastrointestinal tract. The prothrombin time was not sufficiently low to account for bleeding. According to Bockus,[1] 25 per cent of the patients with carcinoma of the pancreas have guaiac-positive stools. Some of them, of course, undoubtedly have low prothrombin concentrations.

The albumin was definitely low and almost down to the range at which hypoalbuminemic edema develops. Perhaps part of the ascites and dependent edema was due to low-borderline albumin in conjunction with other factors. The low albumin is suggestive of cirrhosis, but it does not prove long-standing liver disease.

The prothrombin time was not quite normal but was not very low. It may have been due to liver disease and probably was. The slightly high alkaline phosphatase is no help in deciding which kind of liver disease he had. Forty per cent of the cases[2] with widespread metastases in the liver and three quarters of those with cirrhosis may show phosphatase determinations below 10 units. A paracentesis yielded almost 4000 cc. of fluid. The fluid, with a specific gravity of 1.006, was probably a transudate. It was not grossly hemorrhagic. There was a preponderance of white cells over red cells, but these could easily have been epithelial cells, carcinoma cells or lymphocytes. This sounds more like massive peritoneal transplants of carcinoma than like a cirrhotic transudate. A second paracentesis performed four days later was similar to the first. The low specific-gravity reading of 1.004 was undoubtedly an erroneous figure, as the specific gravity rarely drops below 1.005 at the very lowest.

The disease was progressive, and a peritoneoscopy was performed to establish a diagnosis. After the peritoneoscopy the patient went into what sounds like clinical shock and over a period of several hours lapsed into a state that resembled cholemia, in that he had acidotic respirations and became more jaundiced, confused and restless. He died in coma on the thirteenth hospital day.

There was no clinical evidence of bleeding esophageal varices. That does not rule out bleeding, because if the bleeding was only moderate and the patient was not eating, the blood might have remained in the gastrointestinal tract for several days before it could have been detected. A serious intrahepatic disease to be considered is Laennec's cirrhosis. There are many points for this in the history, but there are also many points against it. The points against cirrhosis are the absence of spider angiomas, the absence of liver pain, the lack of evidence of external collateral circulation, the absence of a sore raw red tongue, and a spleen that was not palpable at any time except possibly immediately before death. These should be present with cirrhosis of a sufficient degree to cause death from hepatic failure within a few days. If spider angiomas were present they would not rule out metastatic disease of the liver. You may recall a clinicopathological case[3] presented a little over a year ago with probably normal liver cells and massive metastases from oat-cell carcinoma of the lung. That patient had spider angiomas and even gynecomastia, so these are not specific signs for Laennec's cirrhosis. There is not enough evidence to say this was a typical Laennec cirrhosis.

I might mention hepatoma, which usually develops only in patients with cirrhosis and may lead to a rapid downhill course. There were not too many signs to suggest hepatoma. A diagnosis of cirrhosis had not been established. Toxic cirrhosis I do not believe has any basis in fact. Lead as a toxic agent is only a weak possibility; exposure to carbon tetrachloride was only questionable. Infectious hepatitis is ruled out because of the patient's course and a number of features of the disease.

Another possibility is a portal-vein thrombosis, which does not necessarily lead to a dramatic exitus or even to the immediate development of dramatic signs. I do not believe that that was the primary trouble in this particular patient, because the history was not too suggestive. Hepatic-vein thrombosis or Chiari's syndrome could have been present, it is true, but I would expect to have been told about a larger liver and more tenderness; apparently the size of the liver could not be determined accurately because of the ascites. I would doubt very much if he had Chiari's syndrome.

A sixth possibility is metastatic disease of the liver, and many points in the history and the examination point to it. The liver did enlarge, as determined by both x-ray and physical examinations, and the course was progressively downhill.

I shall settle for metastatic disease of the liver as the primary diagnosis.

Then the question of the source of the carcinoma arises. In one large series of males[4] the source of extensive metastatic disease of the liver was the stomach in 26 per cent of the cases. I do not believe there was enough evidence for that in this case. About 14 per cent were from the genitourinary tract; in this case there was no evidence whatsoever of that. About 20 per cent were from the small or large bowel; the only evidence here was the gas pains after eating, which could indicate partial bowel obstruction. The symptoms were not suggestive of gastrointestinal-tract carcinoma. Fourteen per cent of cases in the series that I have mentioned were from other parts of the gastrointestinal tract, — that is, the pancreas, the gall bladder, the bile ducts and so forth. I believe that was the origin of this patient's metastatic malignant disease.

If there was carcinoma of the gall bladder there would probably have been evidence of total obstruction, because the carcinoma would metastasize not only to the liver but also to the porta hepatis, involving the biliary duct system. This patient obviously did not have complete obstruction. That does not rule out duct involvement. In the absence of obvious gallstones and without a well defined history of gall-bladder disease, I have some difficulty in making that diagnosis. The pancreas is a good possibility. In this particular case the signs did not point unequivocally to carcinoma of the pancreas, as the patient did not complain of back pain. However, the pain in pancreatic carcinoma may have many bizarre patterns; only three fourths of these patients do have pain. The lungs are the primary site in 5 per cent of the cases of metastatic disease of the liver. There was no evidence of that in this case, although a small lesion could have been hidden. There was never a dramatic change in the pulmonary symptoms.

I shall settle on metastatic disease of the liver as the primary diagnosis, with the pancreas as the most likely source and with the gall bladder as a less likely site. It is difficult to explain the varices with that diagnosis. It is not uncommon for carcinoma of the tail and body of the pancreas to invade the splenic vein or to cause thrombosis of the splenic vein, in which case a rise in the splenic-vein pressure might produce a change in the size of the spleen and the esophageal varices. Also, it is unusual but possible to have portal hypertension on the basis of simple metastatic cancer of the liver without compression of the portal system. I suspect that the patient may have had splenic-vein thrombosis or invasion by tumor and possibly some degree of liver fibrosis, but I do not believe that was the primary disease or the cause of death. The patient undoubtedly died in cholemia, which was probably precipitated by bleeding after the peritoneoscopy. In addition to the metastatic disease of the liver, I shall say that he had blood in the peritoneal cavity from a bleeding biopsy site. He obviously had hiatus hernia and emphysema.

DR. EDWARD B. BENEDICT: Dr. Point, would you say that metastatic carcinoma would give rise to so much ascites?

DR. POINT: In pancreatic carcinoma 10 to 20 per cent of the cases may well do that. That would be particularly so if there were any thromboses in the portal vein or significant hypoalbuminemia.

DR. HANELIN: Is that without peritoneal implants?

DR. POINT: I cannot answer that question.

DR. BENEDICT: I believe that so much fluid is usually against metastatic carcinoma of the liver alone.

DR. POINT: He may have had cirrhosis also.

### CLINICAL DIAGNOSIS

Cirrhosis of liver with esophageal varices.
? Carcinoma of liver.

### DR. POINT'S DIAGNOSES

Metastatic carcinoma of the liver from pancreas, ? gall bladder.
Splenic-vein thrombosis or invasion of vein by tumor.
Hemorrhage into peritoneal cavity.
Hiatus hernia.

### ANATOMICAL DIAGNOSES

*Cholangioma, with metastases to peritoneum and regional lymph nodes.*
*Cirrhosis of liver, portal type.*
Ascites.
Esophageal varices.

### PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: Dr. Benedict, you looked into this man's peritoneal cavity through

your peritoneoscope and took biopsies. Will you tell us your impressions?

DR. BENEDICT: I made a preoperative diagnosis of cirrhosis with a question of carcinoma of the liver. After peritoneoscopy we decided that he had cirrhosis of the liver, hepatoma and carcinomatosis, on the basis that the liver presented an irregular edge and was stony hard and somewhat hobnailed. In addition, we found multiple peritoneal implants on the round ligament, the liver, the abdominal wall, the bowel and the omentum and in the peritoneum of the pelvis. We all believed that the nodules on the surface of the liver looked like peritoneal implants rather than metastatic carcinoma within the liver. We took a biopsy from the liver and from the abdominal wall.

DR. MALLORY: Both the liver and the peritoneal specimens showed adenocarcinoma essentially similar in appearance.

At autopsy we found a large, very firm, nodular liver that contained many multicentric foci of obvious tumor and also appeared to show an underlying cirrhotic process rather coarsely nodular in type. The liver did not have the pale yellow color and the greasy consistency that are characteristic of Laennec's cirrhosis and are seen so commonly in patients with chronic alcoholism. Few of our cases of Laennec's cirrhosis of the liver are associated with a large intake of beer, as in this case, but usually with an intake of distilled liquors of some type. At the time of autopsy the prosector was a little uncertain about the presence of varices but fortunately put through a section for microscopical examination across the entire lower esophagus, which showed multiple though relatively small varices. The peritoneal cavity contained a moderate amount of pinkish serous fluid. There was certainly no evidence of massive hemorrhage into the peritoneal cavity to account for the development of clinical shock. When the intestinal tract was opened small amounts of black, tarry fecal material were found in the lower bowel, indicating that there had been at least some degree of hemorrhage within the gastrointestinal tract. This, however, hardly seemed adequate in quantity to explain the development of shock. One very surprising feature was the small size of the spleen, which weighed only 150 gm. In a patient with cirrhosis and esophageal varices one would rather confidently expect considerable enlargement of the spleen, at least up to 350 to 400 gm., — possibly even larger than that. There were numerous small peritoneal implants of tumor.

DR. BENEDICT: Isn't that unusual in hepatoma?

DR. MALLORY: We did not make a diagnosis of hepatoma on the biopsy; we made a diagnosis of adenocarcinoma. Now that all the evidence is in, I think we can quite confidently call this an intrahepatic cholangioma. Primary tumors of the liver can be of two types. In one, the cells differentiate to resemble liver cells and may even carry out some of the physiologic activity of liver cells, such as the excretion of bile. In the second type the cells differentiate to resemble the cells of the cholangioles or small bile ducts. The appearance, however, is not sufficiently distinctive to enable one to distinguish it from metastatic adenocarcinoma arising in the pancreas or in some portion of the gastrointestinal tract. It is only after thorough postmortem examination and the elimination of a possible foci of primary tumor that one can be sure of the diagnosis of cholangioma. Another point that I think is helpful is that cholangiomatous growths have the power of stimulating the production of very large amounts of fibrous stroma. The tumors are almost always markedly scirrhous. This is in sharp contrast to hepatomas, the liver-cell type of tumor, in which stroma is minimal.

DR. BENEDICT: Was death due to hepatic failure instead of to these other things?

DR. MALLORY: I can only infer that. Unfortunately, we did not make an examination of the head, but a prolonged coma is very characteristic of death from liver insufficiency and may occur in the complete absence of any reduction of bilirubin or of bile salts.

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## "THE GOOD OLD DAYS"

THE establishment of good public relations for the medical profession and the development of an understanding and sympathetic press may at times seem discouraging even in these enlightened days. Comprehension of medical ethics and medical etiquette by lay persons is not always completely free from confusion, nor are all elements in the profession itself entirely co-operative in the attempts that are being made to ensure for the public only the safest and the most enlightened care available. These topics are no less important now for having been mentioned before.

When one compares the integrity of the service that is offered today, however, with the standards that currently prevailed less than ninety years ago, one is impressed by the probability that there is as yet no occasion for despair.

In this respect the pages of a leading Boston newspaper for Saturday morning, November 3, 1866, offer an interesting contrast with the efforts that the same paper, with most others, is now making to interpret correctly to its readers the progress that medicine is making in both science and service.

The four-page publication of 1866 presented with no apparent hesitation the advertisements of sixteen professed physicians desirous of making known to the public the sort of services that they were ready to dispense. Most of them appeared on the front page. Prominent among these was the "Ladies' Notice" of Dr. A. H. Hayes, "Professor of the Diseases of Women, and Vice President of the American College of Physicians," who was able to treat "with perfect success all Female Complaints" and indeed had already treated more of these cases than any other physician in the country. Furthermore, it was well known (or so he stated) "that those who place themselves under his care can religiously confide in his honor as a gentleman, and can confidentially rely upon his skill as a physician."

But that was for the weaker sex. Their gentlemen friends, —

> young men, who, by indulging in secret habits, have contracted that soul-subduing, mind-prostrating, body-destroying vice — one which fills our lunatic asylum, and fills to repletion the wards of our hospitals —

were exhorted to consult Dr. H. without delay, for no mercury was employed and a sure cure was warranted.

A Dr. Hill, in an even more outspoken manner, guaranteed a cure in all cases of Gleet and Venereal Diseases of from one to ten years' standing; his competitor, Dr. Morrill, in offering relief to those afflicted with Stricture of the Urethra, warned his prospective clients against

> some who call themselves Doctors, and advertise that they have been in practice for many years, when in fact they are ignorant of the first principles of the Healing Art.

Dr. Morrill, it happened, had given "nearly TWENTY-FIVE years to the study of Private Disease and Female Complaints," during the last sixteen of which he had

> effected the most wonderful cures upon record, and given his medicines and relief to more than seventy-five thousand

cases of Gonorrhoea, Gleet, Syphilis, and Venereal Affections, as well as those of Impotency, General Debility, Fluor Albus, Irregularities and all manner of kindred complaints.

Perhaps the most candid of these gentry, however, was Dr. C. H. Sholes, a student of private diseases and female complaints, and the proprietor of a hospital, who devoted much of the time that he was able to allot to young men

to the treatment of those cases caused by a secret habit which ruins both mind and body, unfitting the unfortunate individual for either business or society. The sad effects of these early habits, or the excess of riper years, is to weaken and debilitate the constitution, destroy the physical and mental powers, diminish and enfeeble the natural feelings, and exhaust the vital energies of manhood; . . .

Dr. Sholes had his own natural feelings, and of the finer sort, too, for he thus addressed himself "to the People of New England":

Knowing the prejudice that exists in an intelligent community against the Physicians who advertise, the public will please discriminate for themselves between the plain statement of what disease and misfortune require, and the vaunting egotism that disgusts every one who reads the fictitious bragadocio and forged certificates that make the bulk of medical advertisements.

I am, very respectfully, your ob't serv't,
C. H Sholes

The stock of the Puritans had not died in the first post-war year of 1866, but it was certainly at a low ebb!

## READABLE MEDICAL WRITING

Rudolph Flesch, author of *The Art of Readable Writing*, advises, in effect: Make it a story; at least make it personal; keep words and sentences simple and short. As a consultant of the Associated Press News Service and a number of publishers, educational and governmental agencies and corporations, he should know. He quotes, in fact, line and book from Shakespeare to Mencken to justify sentence fragments, split infinitives and dangling participles. To be read easily and retained, the written text must sound like conversation; it goes without saying that the writer must have something to say.

Medical writers frequently have a message, but too many medical readers look at the title and scan the summary, reminding one of Groucho Marx's famous scene in which he dictated a letter. He gave his secretary an elaborate salutation and a flowery closing sentence. When she asked for the rest of the letter he replied: "Never mind, the body will be shipped later." Medical literature is filled with dead bodies. Case reports consist of a six-figure hospital number followed by stereotyped civil and occupational data. Patients are too often only cases when they might be more interestingly considered as persons. The characterizations of old-school clinicians, combining medical fact with personal drama, made facts stick.

This is not to advocate "slick" writing for medical journals but to remind authors that medical writing is an art that has its own technics, which must be acquired and developed. The purpose is the communication of medical facts and interpretations. The writer and the reader have reciprocal functions. Flesch would say: Without readability there is no reader. Medical authors may well study Flesch's suggestions. If some of them are put in practice that part of the medical public may be recaptured that now depends for medical information on detail men and the shining brochures of the drug companies.

## ANATUBERCULIN VACCINATION

Convincing proof of the success or failure of protecting children against tuberculosis by vaccination has been difficult because of the lack of exact clinical experiments; for intentional exposure of a child or group of children to tuberculosis is not in keeping with modern ethical codes. However, in Italy, during the stress of the last world war, an unintentional clinical experiment took place and has been recently reported in an Italian pediatric publication.*

In Italy, Petragnini's anatuberculin (killed tubercle bacilli), instead of B.C.G., is the prevailing means of vaccinating children against tuberculosis.

During the war years, an overcrowded institution for babies near Venice was the site of an epidemic of active tuberculosis. The massive exposure was believed to be due to the admission of babies with active tuberculosis and to their being left in close contact with other babies in small rooms. Of 134 babies in the institution, 54 had been vac-

cinated with Petragnini's anatuberculin; 78 were unvaccinated. Of the 54 vaccinated infants, 9 had developed within one year a tuberculous infection, all being of the hilum glands; there were no deaths. Of the 78 nonvaccinated infants, 21 developed tuberculosis, 9 having hilar lymphadenitis and the other 12 exudative lesions—acute miliary, meningeal or active pulmonary; 11 died.

The epidemic had brought about an unintentional but quite conclusive experiment, which indicates the efficacy of Petragnini's anatuberculin in protecting infants against tuberculosis. Even infants who received the prophylactic injections as short a time as one month prior to the epidemic appeared to receive protection.

*Ferro, A., and Taronna, S. Esperimento Clinico, Involontario, sulla Resistenza di Bambini Vaccinati e Non-vaccinati contro la T.B.C. di Fronte a Contagio Altamente Infettante. *Clin. Pediat.* 32:225, 1950, and 33:1, 1951.

## FESTIVALS OF BRITAIN

THE word "festival" is a felicitous one in the English language, implying as it does "revelry" or "a time of feasting or celebration." Its use may be at times confusing, however, as in Great Britain this summer where, in addition to the ubiquitous Festival of Britain, other sections, not expected by the uninitiated, have joined in the symphony. Such are the annual festival of music in Edinburgh and the Charles Hastings Memorial Festival recently held at Worcester for a special medical purpose.

"Oh, to be in England," as Browning nostalgically expressed it, "Now that April's there!"

The Charles Hastings Memorial Festival was held in Worcester, not in April but on May 30, — a day of perfect weather, as the accounts all emphasized, — to honor the memory of the founder of the British Medical Association, Sir Charles Hastings, who in 1832 inaugurated at the Worcester Royal Infirmary the Provincial Medical and Surgical Association, from which the British Medical Association developed.

The celebration was carried out with characteristic circumstance, though without that pomp that has come to represent an excessive degree of circumstance. "Doctors in academic robes, entering the Cathedral by the west door from the terrace overlooking the river Severn," according to the *Lancet's* account, "took part in the special service, conducted by the Dean, the Very Reverend W. E. Beck, during which Sir Henry Cohen, president of the Association, laid a wreath by the Hastings memorial window."

After the service a luncheon was served in the banqueting room of the Guildhall, where the first Charles Hastings Memorial Lecture was delivered by Dr. W. H. McMenemey, who gave in brief Sir Charles's history and that of the founding of the association.

Perhaps not entirely by chance, the Hastings Festival preceded the opening of the medical program of the Festival of Britain by less than a week. This series of symposia that lasted well into July was arranged jointly by the Royal Society of Medicine and the British Medical Association. It was opened in the Barnes Hall of the Society by the Earl of Athlone, with Lord Webb-Johnson presiding.

"It is a happy augury for the future," as the *British Medical Journal* expressed it, "that the Royal Society of Medicine and the British Medical Association have made this first and successful attempt at co-operation in what matters to them both — the advance in the science and practice of medicine."

*The Pictou (N. S.) Chronicle mentions the death, at East River, of Mr. John Chisholm, of dropsy. He had been tapped 228 times, by which the enormous quantity of 358 gallons of water were taken from his body.*

Boston M. & S. J., *August 13, 1851*

## OBITUARY

### JOHN FALLON

1901–1951

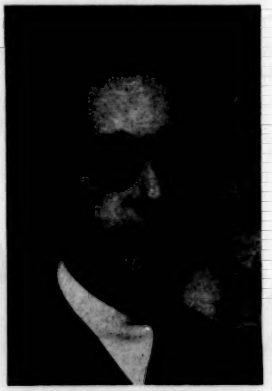
John Fallon was born on September 17, 1901, in Worcester, Massachusetts, the son of Dr. Michael F. and Ellen (Ford) Fallon. His father, a well known surgeon of Worcester County, had been surgeon-in-chief of St. Vincent Hospital, in which capacity John succeeded him, and was the founder of the Fallon Clinic, of which the son was director at the time of his death on June 21, 1951.

John's early life was spent in Worcester, where he attended public and parochial schools and took his

bachelor's degree at Holy Cross College in 1919.

He graduated from Harvard Medical School in 1923, served internships at the St. Vincent and Peter Bent Brigham hospitals and became a fellow of the Mayo Clinic, after which he settled in Worcester for his life work in surgery.

His first wife, Kathleen, sister of Dr. James C. McCann, died after the birth of her second son, John, the surviving child of this marriage. Dr.



Dr. John Fallon

Fallon later married Frances H. Lohbauer of Madison, Wisconsin, who, with six children, survives him.

In his active but tragically brief life John Fallon had been a councilor of the Massachusetts Medical Society since 1937, a member of the Committee on Publications since 1944, a trustee of the Boston Medical Library from 1946 until the time of his death, president of the New England Obstetrical and Gynecological Society from 1947 to 1948, a delegate to the American Medical Association and president of the Alumni Association of the Mayo Foundation. In 1942 he received the honorary degree of Doctor of Science from Holy Cross College. He was orator of the Worcester District Medical Society in 1948 and at the time of his death was orator-elect of the Massachusetts Medical Society.

A fellow of the American College of Surgeons, he also held membership in many local, sectional and national societies, including the American Association of the History of Medicine, the Medical Library Association and the Biological Photographic Association.

His interests were varied and genuine, his work of a high order and his friendships deep and lasting. His wise counsels will be missed by those many who have benefited from them.

## MASSACHUSETTS MEDICAL SOCIETY

### DEATHS

Bresnick — Nathan T. Bresnick, M.D., of Mattapan, died on July 10. He was in his forty-eighth year.

Dr. Bresnick graduated in 1927 from Tufts College Medical School, at which he was an instructor in medicine at the time of his death.

His widow and two sons survive.

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Fenton — Alfred A. Fenton, M.D., of Norwood, died on May 5. He was in his sixty-seventh year.

Dr. Fenton received his degree from the University of Vermont College of Medicine in 1908. He served as president and secretary of the staff of Norwood Hospital, assistant superintendent of the Massachusetts Hospital for Crippled Children and school physician for the Norwood schools. He was a fellow of the American Medical Association.

His widow, two sisters, a niece and a nephew survive.

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## MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

### TRADITIONS, ATTITUDES AND ALCOHOLISM

Traditional popular attitudes in regard to alcoholism emphasize primarily the act and frequency of drinking and the reactions to the quantity consumed. Rarely is there any consideration of *why* an individual may take to alcohol and lose control over its use. Apparently tradition has settled this point in the minds of many by the implication that alcoholism is a condition brought about by lack or perversion of will power, by dementia or by some hereditary taint. It is true that some who appear to be alcoholics are psychotic to some degree or possess a serious character disorder. However, the majority, particularly those in the early stages, are striving to attain and maintain accepted standards of responsibility and ethics.

The prime requisite for recovery on the part of the alcoholic is to acknowledge and accept wholeheartedly and unreservedly his inability to use alcohol with control. This process, which has been variously identified as a "conversion," an "act of surrender" or a "gaining of insight," is merely the breaking down of an alcoholic's resistance to accept the demands of reality. This resistance is fostered by alcohol, which satisfies in fantasy the alcoholic's need for obliteration of pain or unhappiness. Moreover, this resistance is reinforced by traditional attitudes that label the alcoholic as a deteriorated, weak-willed, ungrateful egotist, destroying himself and bringing unhappiness to loved ones.

In acknowledging and accepting his inability to use alcohol with control, the alcoholic takes an essential step toward giving up what has become imperceptibly and unconsciously a major solace in

life, even though destructive. However, the alcoholic believes that if he takes this step he admits also to everything that traditional attitudes toward alcoholism imply. This bolsters his resistance to efforts at cure and deters him from seeking aid and from co-operating genuinely with efforts directed at his recovery.

If the alcoholic is to be helped, traditional attitudes must change and conform to the scientific fact that alcoholism is an *illness*. Medicine with its allied professions and institutions should discard its traditional ostrich-like attitude and assume the leadership in bringing this about by example. Services and facilities of high caliber should be made available to alcoholics in all stages and states. Moreover, energetic research should be directed toward uncovering reliable and unbiased scientific data concerning etiology, diagnosis and therapy. In this way a great contribution can be made to the progress of science and the welfare of society.

JOHN C. AYRES, M.D., Director
Division of Alcoholism

## CORRESPONDENCE

### INFORMATION WANTED

*To the Editor*: On January 19, 1951, Danny Matson, of 70 Cranch Street, Quincy, Massachusetts, disappeared from his home. An extensive search was made at that time and still continues.

Danny, when he disappeared, was four years and five months of age, forty inches in height and weighing thirty-five pounds. He has blue eyes, ruddy complexion and sandy-brown hair.

A reward of five hundred dollars has been offered for his return unharmed. Anyone having any information is requested to notify the Quincy Police Department.

I am sure that the medical profession has the deepest sympathy for the sorrowing parents, who believe that Danny is still alive, even though many months have elapsed. It is hoped that through the medium of this letter one of our New England doctors may hear a rumor or find a clue that will enable this child to be restored to his home and parents.

Wollaston, Massachusetts E. MURRAY BRITTON, M.D.

### PUBLIC HEALTH VS. PREVENTIVE MEDICINE

*To the Editor*: With the continued growth of the health-center movement and expansion of public-health facilities into every phase of the practice of medicine, it is advisable to take stock occasionally of one's fundamental concepts. In my own city of Quincy a "health center" has recently been established and, according to a four-column item in the local newspaper, this center "is dedicated to preventive rather than curative health. It does not overlap into the field of the practicing physician." The article goes on to say that the general rule is this: "If you think you are sick, go to your doctor and find out; if you think you are well, but want to keep a check on yourself, go to the health center for certain types of examination."

To me this philosophy sounds both foreign and obnoxious. The practicing physician practices *medicine*. He practices both *curative* and *preventive* medicine. Whether the patient desires a routine checkup because he is well or desires an examination because he is sick, it is the physician and not the health center that he should be advised to consult.

Every physician practices preventive medicine in his daily work. The dermatologist who removes a precancerous lesion of the skin practices preventive medicine. The radiologist who makes a diagnosis of tuberculosis practices preventive medicine. The surgeon who removes a gall bladder with stones before the patient develops gallstone ileus or gallstone pains is practicing preventive medicine. In every field the practicing physician practices preventive medicine.

There is a confusion of ideas that I believe deserves to be straightened out — and straightened out definitely. *Public health and preventive medicine are not one and the same thing.* The function of the health center is to serve public health. It is to provide the physician with a repository of vaccines, sputum bottles and Wassermann tubes that he can use when he needs them. It is to provide offices for sanitation, food inspection and plumbing. It should provide comfortable office space for the Red Cross and the visiting nurses. It should, however, never delude itself with the idea that it is a place in which medicine might be practiced — either preventive or curative.

Quincy, Massachusetts JOSEPH D. WASSERSUG, M.D.

## BOOK REVIEWS

*An Introduction to Pathology*. By G. Payling Wright, D.M., F.R.C.P. 8°, cloth, 569 pp., with illustrations. London: Longmans Green and Company, 1950. $6.00.

When one sees the word "Introduction" in a title, one ordinarily assumes, at least in the United States, that the subject matter deals with the simpler and uncomplicated ideas and methods of a subject. In medicine, it often refers to books for nurses. In Dr. Wright's distinguished "Introduction to Pathology" this is certainly not the case. This book presents a very thorough and excellent dircussion of the general principles of pathology and the causes of disease. It is a preparation for so-called systemic pathology and clinical medicine, and therefore the word "Introduction" in the title is literally correct.

In the teaching of the preclinical sciences there has been a tendency in recent years to introduce more and more of the clinic, with practical applications. It's easier for the student to remember more about conditions if you show him a patient with that same condition. All this is certainly true, but often these dramatic demonstrations are "gained at the expense of an adequate understanding of the mechanisms that underlie disease processes."

This book does not deal primarily with morbid anatomy and morphology. In fact, the author states that "the era in which pathology was primarily a branch of morphology, and studied with the same technical methods, has passed." This fits his definition of pathology as "the science concerned with abnormal states of the body, the functional disorders that accompany them, and the causes that bring them about." Thus, throughout this book there is emphasis on etiology and on the functional approach, — chemical and physiological, — to the fundamental disease processes such as inflammation, infections, including immunity, tissue repair, edema and thrombosis. The chapter on thrombosis is outstanding.

The functional approach is evidenced by the large numbers of graphs, charts and tables that are scattered throughout the chapters. These do not in any way detract from the excellent gross photographs and photomicrographs. The method of numbering the figures by the chapter number followed by the number of the figure within the chapter is undoubtedly helpful to the author and the publisher but of little value to the reader. For example, Figure 16.14 is the 14th figure in chapter 16. Moreover, this photograph of multiple pulmonary infarcts looks like one of septic infarcts rather than of the usual bland infarcts that are described in the text.

The references are numerous and very well selected, including both those of the original descriptions of a condition or experiment and those of very recent chemical and physiologic studies.

The last eight chapters, about one fifth of the book, deal with the general principles of neoplasia. The chapter on the "precancerous state" might be improved by more photomicrographs. The last chapter on the pathology of some of the more common tumors is much too short and really does not belong in this book.

As general pathology is taught in the United States at the beginning of the student's second year, the book would probably be too advanced. This book is based on Dr. Wright's lectures to students just before beginning their clinical work

— in other words, at a time that would correspond to the end of the second year or the beginning of the third. The material is presented in so scholarly a fashion and so thoroughly, with so much emphasis on function, that I should recommend it to every medical student for study prior to his courses in clinical medicine.

B. Castleman, M. D.

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*Surgery of the Shoulder.* By A. F. DePalma, M.D. 4°, cloth, 438 pp., with 454 illustrations. Philadelphia: J. B. Lippincott Company, 1950. $17.50.

The human shoulder joint, because of its anatomic structure, is peculiarly susceptible to injury. Recovery from such disability is often very slow. Exact diagnosis of injuries is difficult, as is also the prescribing of effective treatment. For these reasons a book on the surgery of the shoulder is most welcome. DePalma labels this book "Surgery of the Shoulder," and the surgery of lesions in this region is effectively covered. However, the book could equally well be called "Lesions of the Shoulder," since all the disabling conditions of the shoulder joint are described. The comparative anatomy of the forelimb is first reviewed, with the evolution of the arm. After a discussion of the surgical anatomy the author discusses with studied brevity and completeness the various injuries and other disabilities centering about the shoulder. The greatest contribution of this book is the study of the progressive changes in the shoulder in advancing decades. From a study of a large number of shoulders studied before death, with careful post-mortem dissection as well as histologic examination, he shows that many so-called lesions are the common attritional changes that appear as the person grows older. He also shows that many of these lesions, which previously were routinely subjected to extensive surgical procedures, respond to patient and persistent physiotherapy. Although anatomic integrity is not regained, good function is often recovered. Even though the text is often dogmatic, one can forgive the author, since he speaks from experience. This book represents a tremendous amount of original work. It is a worthy companion volume to Codman's earlier book on the shoulder. It is well written and well illustrated. There is a good bibliography. This book is recommended to all orthopedic and industrial surgeons.

J. G. Kuhns, M.D.

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## BOOKS RECEIVED

**The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.**

*Favorite Prescriptions.* Edited by Sir Heneage Ogilvie, K.B.E., D.M., M.Ch., F.R.C.S., and William A. R. Thomson, M.D. 8°, paper, 76 pp. London: The Practitioner, 1950. 4s.

In this small collected book Lord Horder discusses prescriptions in general, and 8 British physicians present their favorite prescriptions used in general practice and in diseases of the lungs, nervous system, skin, eye, ear, nose, and throat, and in surgery and physical medicine. There is a good index, and the book is well published.

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*Pain and Its Problems.* Edited by Sir Heneage Ogilvie, K.B.E., D.M., M.Ch., F.R.C.S., and William A. R. Thomson, M.D. 8°, cloth, 194 pp. London: Eyre and Spottiswoods, 1950. 12/6. (The Practitioner Handbooks.)

This composite book is the joint work of 18 contributors. The general chapters discuss physiology, clinical aspects, meaning and measurement of pain, and pain of nonorganic origin, its relief in childhood and the principles of treatment. The other chapters deal with the various types of organic pain. The material is well arranged. There is a good index, and the book is well published. It should be in all collections on clinical medicine.

*Emotions and Memory.* By David Rapaport, Ph.D., research associate, Austen Riggs Foundation. Second unaltered edition. 8°, cloth, 282 pp. New York: International Universities Press, Incorporated, 1950. $4.00. (Menninger Clinic Monograph Series No. 2.)

This volume is an unaltered reprint of the original edition published in 1942. Dr. Rapaport, after due deliberation, preferred to reissue his text without alteration because he believes that interest in the subject is greater than ever, and that "the sections on the theory of emotions, the psychoanalytic theory of memory, and the psychopathology of memory are, in spite of many limitations, still the sole systematic treatment of these fields."

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## NOTICES

### ANNOUNCEMENT

Dr. Wilma A. McVey announces the opening of her office for the practice of internal medicine at 106 Warren Street, Needham.

### MEDICO-MILITARY SYMPOSIUM

To keep military reserve medical officers of the Armed Forces, — Army, Navy and Air Force, — posted on the latest developments in the field of medical science, the Second Annual Medico-Military Symposium will be held at the U. S. Naval Hospital, Philadelphia, Pennsylvania,, October 22-27.

Commodore Richard A. Kern, MC, USNR, professor of medicine at Temple University School of Medicine and chairman of the Symposium General Committee, will preside over the opening session.

It is urged that officers make hotel reservations well in advance, since no government housing facilities will be available. The final session of the symposium will be held on Saturday morning, October 27, leaving the afternoon free for officers to attend the Penn-Navy football game.

### NEW ARMY POSTGRADUATE PROGRAM

Two courses in internal medicine are included in a new series of postgraduate short courses for armed-forces medical officers. This new in-service clinical-training program is designed primarily to afford medical officers of the armed services an opportunity to receive the excellent training available in Army installations. A total of 18 courses in 9 medical and 1 dental specialty fields will be given at 5 Army hospitals, the Armed Forces Institute of Pathology, Washington, D. C., and the Medical Service Graduate School, Army Medical Center, Washington, D. C., during the last six months of this year. Other courses will be given in the second half of the fiscal year 1952. (Naval and air force medical officers have been invited to attend these courses.)

The first of the two internal medicine courses, "Present Trends in Internal Medicine", will be given at Walter Reed Army Hospital, Washington, D. C., October 8–12. Attendance will be limited to personnel from the 1st, 2nd and 3rd Army Areas and the Military District of Washington.

The second of the two courses, "Recent Advances in Internal Medicine," will be held at Letterman Army Hospital, San Francisco, California, November 5–9. Attendance will be limited to personnel from the 4th, 5th and 6th Army Areas.

Applications from medical officers on active duty, who will be given priority in the allotment of spaces, will be forwarded through channels to: The Office of The Surgeon General, Department of the Army, Attention: Personnel Division, Washington 25, D. C. Applications from Air Force and Naval medical officers should be forwarded through regular channels. Applications from medical officers not on active duty and from civilian physicians should be forwarded through channels to the commander of the installation offering the course. The commander will select those to attend on a space-available basis. Army medical officers enrolled in the residency training program may attend these courses only if they are given at a hospital at which the resident is being trained.

(*Notices concluded on page ix*)